

# THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—35TH YEAR.

SYDNEY, SATURDAY, MAY 15, 1948.

No. 20.

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### DIAGNOSIS OF GASTRIC DISEASE: SHOULD RADIOLOGY OF THE STOMACH BE ABANDONED?<sup>1</sup>

By V. J. KINSELLA,  
Sydney.

For now I see the true old times are dead,  
When every morning brought a noble chance,  
And every chance brought out a noble knight.

The task of offering something of help and of value in the diagnosis of gastric disease is not an easy one. It is perhaps best fulfilled by retracing the surgical pathways which I have trodden over the last few years and going over the varied and sometimes unconnected problems as they presented themselves. We shall profit more from a study of mistakes than from a study of easy successes.

#### The Foundations of Successful Diagnosis in Surgical Disease of the Gastro-Intestinal Tract.

In diagnosis I would stress two methods which stand out, of prime importance. The first is, of course, a painstaking and time-consuming collecting and analysing of the symptoms and signs. This has been stressed so often that I could press the point only at the risk of boring you. But I should like to emphasize, in the history-taking, the skilful use of the leading question. Not only the classical symptoms, but also the minor ones, if not volunteered by the patient, should be actively sought after and specifically inquired after by the clinician; otherwise they may go undiscovered.

The second great essential is not so well known, and it will appear and reappear, an ever-recurring theme in my address. It is the method of clinical and radiological association, not just radiology investigating the patient

in splendid isolation, relieved only by two or three lines of writing from the clinician. It is radiology directed not by the radiologist, but by the clinician, with the precision and thoroughness that the clinical difficulties demand.

#### The Attitude of the Clinician to Radiology.

The radiologist should be looked upon by the clinician as a valued assistant, but as no more than an assistant, in the search for a diagnosis. Too often, to the grave detriment of the patient, he is looked upon as some kind of oracle or final court of appeal, and his first word is too often allowed to finalize the matter.

In order that the best use should be made of radiology, it is essential that the clinician should thoroughly understand its resources and methods. It may be asked, can such knowledge be expected of a clinician? Yes. It must be demanded of him. Though the resources of radiology are rich, the methods are simple, straightforward and not difficult to understand. The commander-in-chief of a combined operation may never in his life have piloted an aircraft, commanded a ship or constructed a bridge. Nevertheless he must understand the possibilities of each arm and he must know how to make the utmost use of it. So it is with the clinician and radiology. It is the clinician who should be acutely aware of the overall position and of the possibilities; it is only he who has had the opportunity for the intense survey of the symptoms and signs which alone leads to this awareness. And it is he who receives that emotional stimulus to determined effort which is provided by personal contact with the stricken individual. Moreover, the most important element in radiological training is the comparison of the shadow findings with the clinical condition of the patient and, above all, with the after-course of the illness and the operative or autopsy findings. In the absence of these controls the deficiencies in routine methods and the errors in radiological interpretation are repeated indefinitely year after year. The radiologist in most instances does not learn the fate of those who months previously had

<sup>1</sup>Based upon addresses given in Brisbane on June 3 and 4, 1947, during the Queensland Post-Graduate Week.

stood behind his fluoroscopic screen. The patient usually goes elsewhere for the terminal skiagrams. The clinician therefore must be radiologist as well, because only then can radiology be used with the utmost effectiveness.

If, therefore, the radiologist attacks the problem and his attack is fruitless, it is for the clinician to know if there are other forms of radiological attack which may prove more fruitful, and it is for him to request the radiologist to use these other methods. A close liaison between clinician and radiologist is thus an absolute necessity, and the patient must often pass backwards and forwards between them, each helping the other, before the clinical and radiological possibilities have been exhausted. As my cases pass in review I hope to make clear by examples the ultimate effectiveness of this combined operation.

#### Hiatus Hernia.

It is important that clinicians should be aware of the condition of hiatus hernia. It may cause no symptoms, but it may simulate gall-bladder disease or gastric ulcer or coronary sclerosis. In addition it may cause an unexplained anaemia through the bleeding of a frictional ulcer. Other conditions may simulate hiatus hernia. These include gastric and oesophageal diverticula, penetrating ulcer, congenital short oesophagus, and dilatation of the lower segment of the oesophagus, perhaps representing the anterior compartment of the ruminant stomach. Eversion of the diaphragm simulates hernia, but differs from it in probably not needing surgical treatment.

Very often these herniae are not revealed at radiological examination, because the herniation may occur only when the patient strains or is placed in the supine, horizontal or head-down position. In other words, steps must be taken similar to those taken by the clinician when he investigates a possible inguinal hernia.

CASE I.—Mr. A.K., aged fifty-seven years, short and stout, complained for some years of pain in the lower sternal region passing through to the back; the pain came on one and a half hours after food and was relieved by powders, but not so well lately. Many radiologists had reported the alimentary tract to be normal. The patient had always been examined in the upright position. A radiologist was requested to use the supine position, and he reported as follows:

In the supine position, and especially in the left lateral position, portion of the cardiac end of the stomach, including the cardiac orifice, passes through the oesophageal hiatus and there is a reflux of the barium to fill the whole of the oesophagus, which is flaccid and dilated. Once filled in this manner, there is vigorous peristalsis which empties the oesophagus and the cardiac pole of the stomach once more returns beneath the diaphragm.

In many other instances hiatus hernia appears on the skiagrams, even when the patient is in the upright position, but is not mentioned in the radiologist's report. Three such examples are shown; two of the patients had penetrating ulcers of the duodenum and a third had gall-stones in addition to their herniae (Figure 1).

#### Carcinoma of the Cardiac End of the Stomach.

Of seven recent consecutive patients with carcinoma of the cardiac end of the stomach all were referred for radiological examination of the stomach, and in six instances the diagnosis was missed. In five of these six there is evidence which shows that the diagnosis was missed because the appropriate posturing of the patients during the radiological examination was omitted. In four of the six the original skiagrams are available for study. In three the skiagrams show deformities which could hardly have escaped recognition if the patient had been examined in the supine position with rotation. In the fourth (case of Mrs. L.) the original skiagrams, in the upright and prone positions, show no abnormality; but two weeks later, when the radiologist was asked to use the supine position, the huge ulcer and filling defect showed plainly. A fuller discussion of the above-mentioned cases, together with relevant skiagrams, is in course of

publication in *The Australian and New Zealand Journal of Surgery*.<sup>1</sup>

Mrs. F.Mc.G., aged fifty-eight years, complained for eight months of symptoms typical of advanced carcinoma of the cardiac end of the stomach or of the caecum—weakness, grave loss of weight, anaemia and repeated melena, with no other gastro-intestinal symptoms. More recently constant severe pain in the left subcostal region indicated the stomach rather than the caecum. The pain suggested that the growth had spread far beyond the stomach wall. A recent complete radiological examination in Brisbane, including the use of a barium meal, revealed no abnormality. The patient was given a tonic, was treated by her relatives as neurotic, and came to Sydney for a holiday. But even a trip to Sydney was ineffective. Soon after her arrival she was admitted to a private hospital because of her weakness. A physician was consulted and a second radiologist reported the stomach as normal. The physician withdrew gastric contents and found "coffee-ground" material. I examined the patient on November 12, 1947. The films had been taken with the patient only in the prone position. But even in these a slight craginess at the upper pole indicated the growth. The radiologist was now recalled and six days later examined the patient in the supine position. The slight craginess now appeared as a massive filling defect under the diaphragm and was reported as a carcinoma. At operation on November 30 the growth was found to be as large as a full-term fetal head. It had arisen in the posterior wall at the fundus, and had infiltrated the pancreas, kidney and spleen. The spleen appeared like a cap upon it.

It is clear that this growth could have been shown radiologically at a much earlier stage by lateral views with the patient supine.

I now find it hard to avoid the belief that the possession of a "negative" radiological report, with an incomplete set of gastric films, is strong evidence of carcinoma of the stomach.

It is of vital importance that the clinician should understand the necessary posturing of the patient, so that he can direct the radiologist to a thorough examination of that part which is indicated by the clinical circumstances.

#### Lesser Curvature Ulcer.

The fact is not generally recognized that a penetrating ulcer on the lesser curvature may not retain the barium meal while the patient is standing and may therefore pass unrecognized. Dr. Templeton, of Chicago, has shown that this applies to duodenal as well as to gastric ulcer.

CASE V.—Mr. M. had low sternal pain, more severe after exertion and after eating, and oedema of the ankles. He was in hospital undergoing treatment for cardiac disease. The radiologist reported "atonic stomach, with no organic lesion" after examining the patient in the standing position only. But a constant, localized, deep tender point in the epigastrium led to operation, which revealed a "letter-box" ulcer of the lesser curvature penetrating the liver. After operation the pain and oedema disappeared.

An examination of the original films (Figure II) shows that the great ulcer niche was not able to retain barium when the patient was standing. The unrecognized niche can be seen with a small air-bubble in its upper part, and a fleck of barium in its lower part, and with radiating streaks of barium, the gastric rugae, pointing towards it. It is almost certain that this ulcer would have been recognized if the patient had been examined in the horizontal position.

This patient also emphasized the overriding importance of the constant localized deep tender point in revealing disease hidden beneath the wall of the abdomen.

CASE VI.—Mr. E.C., aged fifty-nine years, on January 15, 1947, was admitted to hospital on account of haematemesis and pyloric stenosis. On February 11 the radiologist reported pyloric stenosis, due to an ulcer of the pylorus or duodenum. On February 27 operation revealed a large penetrating ulcer of the lesser curvature, well away from the pylorus, not causing stenosis and not revealed in the skiagrams. It is probable that this ulcer could have been revealed if the patient had been placed in the horizontal position. The stenosis was due to a strong band of adhesions associated with a Paul operation carried out fifteen years previously on the transverse colon.

<sup>1</sup> Two more patients have recently come under my care. The score now stands at eight cases missed out of nine, or perhaps even worse, because some patients were missed more than once. The eighth patient's history is typical and worthy of close study.

### Lesser Curvature Carcinoma.

Carcinoma of the lesser curvature is frequently missed radiologically, even when quite advanced. Here again suitable posturing of the patient would greatly reduce the number of diagnostic failures. Three recent examples are given.

CASE VII.—Mrs. R., aged forty-five years, on July 20, 1945, complained of epigastric pain of one month's duration, unrelieved by food. The radiologist reported "small duodenal ulcer". The carcinoma of the lesser curvature was unrecognized. She was treated by her physician, and improved in health. On March 11, 1946, she was referred by her physician on account of a recurrence of the pain. Her nutrition was good and she had lost very little weight. A further X-ray examination revealed an extensive carcinoma of the stomach. At operation the primary growth was found to spread far into the mesenteries.

A reexamination of the first set of six films shows the unrecognized filling defect and deformity of the lesser curvature (Figure III), which would probably have been made still more clear if the patient had been photographed in the supine position with varying degrees of rotation, so that the normal part of the stomach could roll around the edges of the growth and outline it more clearly.

CASE VIII.—Mr. A., aged twenty-five years, on July 21, 1944, was admitted to hospital with pyloric stenosis following the local excision of a lesser curvature "ulcer" some months previously at another hospital. The films (Figure IV), all taken with the patient in the upright posture, well illustrate the fact that this posture may be inadequate to define the lesser curvature. At laparotomy a large, densely adherent mass was found at the site of the old excision, gastrectomy was considered impossible, and gastro-jejunostomy was done. The patient's condition improved, but he relapsed, and a large, hard mass became palpable in the epigastrium.

On February 14, 1946, a further X-ray examination showed that the original lesion on the lesser curvature was a carcinoma, a diagnosis soon confirmed by the course of the illness (Figure V).

It is probable that if the patient had been photographed in the supine position with varying degrees of rotation the nature of the lesion would have been recognized, at least before his second operation.

CASE IX.—Mr. V.D.A., aged thirty-nine years, on October 19, 1944, complained of epigastric pain, nausea and flatulence, of recent onset. He had had epigastric pain many years previously, due to a duodenal ulcer, but his present pain was different. Radiologically the stomach was reported as normal, in spite of a small filling defect high on the lesser curvature, marked in the skiagram by arrows (Figure VI). The patient was examined only in the upright posture. Medical treatment was given.

On August 30, 1946, the symptoms had continued. The second X-ray film of the stomach was reported as showing no abnormality. The patient was examined in the upright position and two films were taken. The films showed that the lesion had extended along the lesser curvature towards the *incisura angularis*. It can be seen as a blurred or velvety outline in this part, sharply limited distally by an angular deformity which is marked with an arrow (Figure VII). The velvety outline is seen better in the original films, but even in the reproductions it can be appreciated by comparing the edge of the lesser curvature shadow on either side of the lowest arrow. The patient was now treated for nervous dyspepsia.

On January 15, 1947, the symptoms had grown worse. The third X-ray examination (with the patient in the upright position only) revealed "some gastritis, but no ulcer". Two films were taken. The films still showed the velvety outline and angular deformity on the lesser curvature (Figure VIII). The patient was now treated for gastritis.

On March 31, 1947, the symptoms had continued. A fourth X-ray examination (with the patient in the upright position only) is reported as showing "a small penetrating ulcer on the lesser curvature of the stomach". The films show that the velvety deformity of the lesser curvature had been replaced by a rough, irregular serration (Figure IX). The neoplasm had now assumed formidable proportions.

If the patient had been adequately postured during this or during his preceding examinations, it is probable that the deformity would have been sufficiently marked to secure recognition by the radiologist. With the patient in the supine position, with varying degrees of rotation, the still flexible normal stomach wall would have bulged upwards around the growth, so making the filling defect more plain.

On May 20, 1947, the symptoms had grown worse. The fifth X-ray examination was now planned. The radiologist was asked to use supine and oblique positions. The irregularities and the filling defect were now noted (Figures X, XI and XII). It was reported that "this may be due to cicatrization from a healed gastric ulcer, but the possibility of carcinoma cannot be overlooked". It should have been known that, apart from hour-glass deformities, the formation of filling defects by a healed gastric ulcer does not occur, so that carcinoma is certainly present. It is unmistakable in the supine position (Figure XII). But more skiagrams were sought, and became available after the inevitable delay.

On June 15 these skiagrams (Figures XIII and XIV) formed the basis of the sixth X-ray report: "A large carcinoma invading, and encircling the stomach in at least its upper three-quarters." The carcinoma was now enormous and could be plainly felt as a large mass extending down below the left costal margin.

In July, 1947, the patient was admitted to Saint Vincent's Hospital. On July 15, by the transthoracic route, a total gastrectomy was performed, together with the resection of an hepatic metastasis, which appeared to be solitary. On August 28 the patient was discharged from hospital. From the beginning his oesophago-jejunostomy has functioned well. Four months after operation he had a voracious appetite, in spite of the absence of his stomach. But his general condition is at present not good, and it is thought that he has unrevealed metastases.

But for radiology this patient's growth would probably have been discovered at laparotomy two years earlier.

### Gastric Cancer and Radiology.

Few practitioners realize the unreliability of present-day radiology of the stomach. The danger to the patient, especially to the sufferer from early cancer, lies in the undeserved trust which is placed in this form of examination. Perhaps, if forewarned of this unreliability, the clinician would better appreciate and cherish his own art, and more of our cancer patients could secure that form of therapy which alone offers hopes of palliation or cure. Gastrectomy for uncomplicated resistant peptic ulcer is an operation of usually minor severity and almost no mortality. But this is withheld from the cancer patient, usually through the advancement of the disease. Too often laparotomy is delayed by radiology. The fault lies partly with the clinician, who forfeits his clinical birth-right for a mess of shadows, and partly with the radiologist, who sees not because he does not look.

Of course the radiologists have a large volume of work to cope with. But the part examination is not a satisfactory solution for this problem. One hundred part examinations are not an acceptable substitute for fifty thoroughly performed. Many of the part examinations need never have been repeated if they had been thorough in the first place. The part examination loses lives without saving time. Bone work could not be expedited by omission of the lateral views.

Some radiologists defend their present methods and refuse to admit possibilities of improvement. They refer to their high percentage of diagnostic accuracy. This percentage is based upon nothing more than a hopeful imagination. No doubt among the diagnostic "successes" are numbered the patients whom I present in this paper.

I have been accused of collecting the errors made by junior radiologists. But most of the patients reported in this communication were examined by seniors, whose teaching and example are the chief obstacles to the progress of the juniors in gastric radiology. But I was much refreshed by one of my radiologist friends, who, with a greater vision, expressed his dissatisfaction with the present position and set about planning for a better tomorrow. "When that which is perfect is come, then that which is in part shall be done away".

### Pyloric Obstruction.

The diagnosis of pyloric obstruction may be recapitulated here because it is not well described in textbooks of medicine. In these only the classical symptoms of advanced cases are stressed—for example, the vomiting of huge quantities of material in which bile never appears, often at the end of the day, the recognition of food taken

some days previously, and the stomach's appearance as a large sac in the skiagram. This represents a terminal picture in which the stomach has given up the struggle and become atonic.

But there is an earlier stage which should be recognized. The patient complains of epigastric fullness after eating, sometimes relieved by vomiting of fairly large quantities of material. The emptying time may not be prolonged beyond six hours. But the observant radiologist may notice that the peristaltic waves are frequent, deep and powerful. The association of this increased motor activity with a normal or delayed emptying time suggests the earlier stages of a pyloric obstruction. When this is noted, special efforts should be made to fill out the pylorus and the first part of the duodenal cap by manipulation under the fluoroscopic screen. If this cannot be done, and if the normal shapes of the pylorus and the duodenal cap are not seen on the screen or the skiagrams, the diagnosis of early obstruction can be taken as confirmed.

It is most important to remember that a diagnosis of pyloric obstruction at any stage should not be accepted unless special efforts have been made, and have failed, to fill out the normal shape of the pylorus and cap and to show these structures upon the fluoroscopic screen and upon the skiagram. If this precaution is not taken, huge dilatation of the stomach with delayed emptying may be erroneously attributed to a pyloric obstruction, when it is in fact due to atonia or to an obstruction of the intestine.

In a study of the skiagrams pyloric obstruction can be excluded if the lower parts of the duodenum and the upper coils of jejunum appear consistently well filled. These parts normally empty so rapidly that they cannot remain well filled if the gastric emptying is delayed. A study of the area in the skiagrams below and to the left of the greater curvature thus becomes an important part of your scrutiny, as is shown in the two cases now to be described.

CASE X.—Miss "X", a female patient, aged forty-five years, was referred to me with a diagnosis of pyloric obstruction, for gastro-enterostomy as soon as possible. She had been vomiting for two weeks large quantities of material, chiefly the food eaten a few hours after meals, sometimes immediately after meals. She had lost over four stone in weight in three years. She had had hæmatemeses in 1945 and in 1946, three during the last five weeks. Twenty-three years earlier laparotomy had been performed for epigastric soreness and vomiting and loss of weight. No ulcer was found and appendicectomy was performed. Since then she had had vomiting attacks which disappeared if she starved. Eight years earlier she had had an abdominal section performed by a gynaecologist, but the details are not clear. In 1945 two electrocardiograms revealed coronary sclerosis. In 1946 a third showed no abnormality. In 1937 a test meal examination revealed hyperchlorhydria; in 1944 a second revealed hypochlorhydria. The patient was a wasted woman with a smooth tongue. On March 21, 1946, the radiologist reported "small four-hour residue, constant deformity of duodenal bulb, some delay in evacuation, large bowel well filled and free from irregularity".

The violent vomiting in this case and the history of hæmatemeses were certainly suggestive of pyloric obstruction. But the vomiting often followed immediately after eating, and the history contains curious inconsistencies, more suggestive of a functional disorder than of organic disease. A study of the skiagrams cast further doubt on the diagnosis. These showed considerable filling of the jejunal coils. The patient was therefore sent for reexamination on April 11, and this time the pylorus and the duodenal cap were seen to be normal. The presence of organic pyloric obstruction was thus disproved. But the radiologist drew attention to the enormous accumulation of gas in the descending colon, which was so dilated that the stomach was pushed to the right. He suggested an obstruction in the lower part of the rectum. But digital and proctoscopic examination showed this part to be normal. A confident diagnosis of hysterical vomiting was now made. It probably accounted for the previous operations. The patient vomited till she nearly died from inanition, then recovered.

CASE XI.—Mrs. L.W., aged forty years, was admitted to hospital on April 8, 1947. She had been vomiting for two weeks large quantities of material, a pint or two at a time. She was wasted and dehydrated.

The first diagnosis, made before the patient's admission to hospital, was one of almost complete pyloric obstruction and she was sent for urgent operation. She

brought with her a radiological report dated March 19, 1947, and skiagrams which appeared to confirm this diagnosis (Figure XV). In these the stomach was so large that the greater curvature reached almost to the pubis, and it contained most of the barium even after many hours.

The vomitus was seen to be richly coloured with bile, and this ruled out the first diagnosis. The patient was sent again to the radiologist (March 27) with the request to try by posturing and palpation to fill out the pylorus and duodenal cap. These were thus shown to be normal, and therefore no pyloric obstruction was present (Figure XVI). It was now thought for a time that some rare "medical" cause or hysteria was the cause of the vomiting. But there was no collateral evidence for these causes. The patient was watched day after day and the skiagrams were repeatedly examined. Finally the inspiration came; the diagnosis was there in the old skiagrams, but it had not been recognized.

Some of the previous films showed barium passing freely through the pylorus, but none in the upper jejunal coils. Traces of barium in the second and third parts of the duodenum showed this to be of much greater calibre than usual. The duodenum could be seen through the outline of the great, fluid-filled stomach. Was the great size due to primary atony? Or did it mean obstruction at the duodeno-jejunal flexure? The stomach was huge and sack-like at times, and in other films it looked normal. The radiologist was asked to examine the upper part of the alimentary tract for the third time (April 16), and to try by posturing and palpation to press the meal through the duodeno-jejunal flexure. He succeeded in confirming an obstruction at this point (Figure XVII), and this is commonly due to carcinoma. The growth was revealed at subsequent laparotomy.

Here are shown two important methods in diagnosis: firstly, the method which I stressed at the beginning of this lecture, the method of associating and checking clinical and radiological data, one against the other; secondly, the importance of repeatedly considering the evidence, repeatedly viewing the skiagrams, and repeatedly puzzling over the patient. Persistence is sometimes rewarded.

It is also clear that radiological signs may be inconsistent, as were the size and appearance of the stomach in this case. This must not be taken as ruling out organic disease.

This case well illustrates my thesis. Radiology must be directed by the clinician, otherwise it may not only fail to discover the diagnosis, but it may even hinder and delay it. It is the clinician who must supply the motive power when, as so often happens, a special effort is needed.

#### Radiological Technique.

A complete radiological examination of the stomach includes fluoroscopy and the taking of skiagrams. The report of the radiologist on what he has seen during fluoroscopy is inadequate. Skiagrams should be taken in adequate numbers. The clinician should not be satisfied with a single film of the stomach or colon, or with pictures of the pyloric end of the stomach but not of the cardiac end. When the urinary tract is examined the urologist is not satisfied with one film; nor is he satisfied with a series which shows the lower calyces of the kidney but shuts out the upper. Fluoroscopy should be carried out and skiagrams taken with the patient standing, and prone, and supine, perhaps with intermediate degrees of tilting. In each of these positions the patient should be rotated so that oblique and lateral views may be obtained. Only in this way can the anterior and posterior walls be brought into profile.<sup>1</sup>

With the patient standing, the entrance of the barium into the stomach (the oesophageal jet) is observed and the mucosal relief pattern is studied. The stomach appears relatively elongated and the air is at the fundus.

The prone position is the best for study of the pyloric end and of the duodenum. The stomach appears much shorter, but the air is still at the fundus.

The supine position is necessary for a study of the cardiac end, which lies in the abdomen so far posteriorly

<sup>1</sup>These facts are well illustrated in diagrams appearing in "The Digestive Tract", by A. E. Barclay, the Cambridge radiologist.

that it is only in the supine posture that it is well filled with barium. The air passes to the pyloric end and the stomach appears shortened, with a relatively broad fundus. This position is necessary also for the complete study of the posterior wall. As the table is tilted backwards the pool of barium lying at the lowest point of the stomach gradually creeps upwards on the posterior wall, like the tide rising on a shelving beach, until it reaches the fundus. This manoeuvre sometimes shows high posterior wall lesions which cannot be shown in any other way (for example, the case of Mrs. L., of the cardiac end series). The prevailing radiological habit of relying on the splashing technique—that is, trying to force barium by hand from the lower to the inaccessible upper pole of the stomach—cannot be too strongly condemned.

If the clinician undertakes to advise patients with gastric complaints, he should accustom himself to recognizing the typical radiological appearance of the stomach in different postures, so that he may know which part of the stomach has been examined and, if necessary, refer the patient back for examination of that part of the stomach which has been neglected.

If there are no skiagrams with the patient in one of the postures described, it means that the patient has either not been examined in this posture or has been only partly examined. Fluoroscopy without skiagrams is only a partial examination. Very often films must be examined repeatedly, day after day, and repeatedly compared with and checked by the clinical findings. As Barclay puts it: "A vital part of the routine is ample time for examining films carefully on a good viewing box." At one time I thought that, even if there were no films taken in the supine position, the patient had at least been fluoroscopically examined in this posture. But I now know that the posturing is neglected, not only in the taking of films, but also in fluoroscopy. The supine position is neglected usually, the prone position frequently. If the clinician finds that no films have been taken with the patient in the supine position—that is, if no film shows air at the pyloric end—he can safely conclude that the fluoroscopic examination in that position has also been omitted.

At this point it is useful to review the remarks of Dr. F. E. Templeton, of the University of Chicago and the Cleveland Clinic. He is an outstanding authority on the X-ray examination of the stomach and a colleague of Schindler and Brunswick. The University of Chicago Press published his book on the subject in 1944. "These methods [the newer X-ray techniques] differ from the old, not merely in the greatly increased emphasis on films but also in the details of the fluoroscopy itself." He divides the fluoroscopic technique into two parts, the routine examination and special procedures. The routine examination includes: Stage I (patient standing, after taking a small amount of barium); Stage II (patient supine, with the use of the Trendelenburg position); Stage III (patient prone); Stage IV (patient standing, with stomach completely filled with barium). In these stages the patient is rotated so that oblique views are obtained. Templeton then details the routine films which are taken to show thoroughly the various parts of the stomach. Over and above this routine for each case special procedures are added for special problems. There is no mention here of the cursory method in which the patient is fluoroscopically examined and one or two films taken in the standing position. "To the examiner, the films not only offer a means of confirming the fluoroscopic observations but frequently record images not seen in the fluoroscopic examination." "It is not uncommon for the roentgenologist to see changes on the films which he failed to see at fluoroscopy." This, he it noted, in spite of the increased efficiency of modern fluoroscopy which Chicago has done so much to foster. "Films form a permanent record available for comparison with previous or subsequent examinations or with the pathologic specimen." (Or, I might add, with the patient and his symptoms and signs.) "The relative infrequency of carcinoma in this region (upper posterior wall) leads to carelessness. The habit of passing, as 'normal', stomachs in which the region is not adequately seen is easily

formed." Figure XVIII shows the stomach of R.McG. (Case XII), who was recently referred to me on account of a carcinoma apparent at the cardiac end of the lesser curvature. This stomach was passed by a radiologist, the patient having been examined fluoroscopically in the upright position and a "normal" report was dictated before the solitary film was developed. After the radiologist had departed the film was developed and the report was recalled by a technician.

For the cardiac end even the routine of Templeton can be improved upon. The lateral view, with patient supine and the central ray horizontal, has not yet been sufficiently exploited; nor has the method of angulation used in dental radiology, which is applicable to the obliquely placed stomach in sthenic patients, in the antero-posterior films taken with the patient in the supine position.

At present I have under my care a patient who had an hiatus hernia and a simple tumour of the stomach. After repair of the hernia and removal of the tumour the patient was sent back to the radiologist for a testing of the adequacy of the repair. The radiologist made no use of muscular straining or of the supine position. It thus became necessary for me to refer the patient again with explicit instructions about the technique of examination. Another patient had hiatus hernia and a massive penetrating ulcer of the duodenum. After gastrectomy she was referred back to the radiologist to see if there was any alteration in the behaviour of the hernia. The radiologist reported: "No evidence of any hernia about the cardio-esophageal junction." A study of the films showed air at the cardiac end in each. There was no supine view. Neither was the supine position used during fluoroscopy. I was informed that it was not considered necessary in this particular radiological department. This may have been incredible were it not for my own knowledge of previous examinations in this department and in the private practice of the radiologist. But I was interested to note in the department a copy of the technicians' "bible", K. C. Clark's "Positioning in Radiography" (1945). This, like other textbooks, included the supine position in its gastric radiology routine. But the radiologist in this hospital had taken it upon himself to omit an important part of the accepted routine. So have the radiologists in many other hospitals and in private practice. The reason for this omission is unknown to me. The effects are all too familiar.

But tragedy does not always follow. Sometimes the prelude is amusing and the ending happy.

CASE XIII.—Mr. A.W. had gastric symptoms, and on September 15, 1947, was fluoroscopically examined by a radiologist and had one film taken in the standing position. The report was as follows: "The stomach is high and hypertonic, and there is a deformity involving the pyloric third, more suggestive of extensive adhesions than of a prepyloric lesion." The duodenal cap was on the left side of the spine, but this was not noted. Next day a second radiologist observed the progress of the meal and noted that the splenic flexure was high. He therefore secured a plain skiagram of the chest, with the patient standing, and reported eventration of the diaphragm, due to a recent blow on the chest, as a possible cause. (Eventration is a harmless condition for which surgical treatment is not indicated.) But the patient came into hospital and his radiological examination was repeated before the surgeon and his class of students. By request the supine position was now used. It showed the stomach passing high into the left side of the chest, all except the cardio-esophageal junction and the pyloric end. These appeared as though tied close to each other by an encircling string. This showed the condition to be diaphragmatic "hernia". The constricting agent was the tear in the diaphragm through which the stomach and colon had "herniated". Such a condition is dangerous and requires early surgical attention. The tear was successfully repaired next day.

#### Conclusion.

Among these patients with gastric disease much morbidity would have been saved, and perhaps some lives, if the clinician had accepted the guidance of his own art. Instead, he was often diverted by radiology. Such experiences suggested to me the title of this paper. But it may be possible to make better use of radiology. This could

be done if the clinician practising gastro-enterology were to understand the simple principles of radiology and the routine of examination and if he were to ensure their adequate application to meet the particular requirements of his patient. The clinician must also realize the limitations of even the most thorough radiological examination and the fact that the sound interpretation of the shadows can be founded only on assiduous clinical and pathological studies, for which the radiologist has no opportunity. In this way the gastro-enterologist will get more out of radiology for his patient. The urologist, orthopaedic surgeon, neuro-surgeon, chest physician, cardiologist and other specialists have already achieved this. Above all, it must be remembered that the omission of an important step in the radiological routine may ensure the patient's doom with as much certainty as may the omission of an important step in operative routine.

In order to stress the frequency of the errors and pitfalls which I have described, I would point out that all the cases were found within the limited field of my own practice, during a brief period of three or four years. Only one patient goes back before this time.

#### Summary.

1. The easier the diagnosis of gastric cancer, the harder the cure.
  2. The first essential in diagnosis of gastric cancer is to suspect it. Either local symptoms (for example, pain or dyspepsia) or general symptoms (for example, weakness and anæmia) should make one suspect it. A painstaking collection and analysis of these, together with the physical signs, remains the basis of diagnosis in gastric diseases.
  3. Gastric cancer cannot be ruled out by "negative" clinical symptoms or signs, nor by the youth of the patient.
  4. It cannot be ruled out by a "negative" radiological report. This is not so well known. "Negative" radiological reports in gastric disorders are not trustworthy and too often sound the death knell for the patient.
  5. It can be ruled out only by the disappearance or by the persistence of the clinical and radiological signs, unchanged in pattern, over a period of at least two years.
  6. A filling defect or ulcer unrevealed under one set of radiological conditions may stand out clearly when conditions are altered.
  7. In no branch of radiology may it be said with more truth than in the radiology of the gastro-intestinal tract that the harvest of early curable lesions is directly proportional to the effort expended. At present patients are not receiving the benefit and protection of an adequate routine. The present-day half-examinations are in the end neither time-saving nor life-saving, but very often the reverse.
  8. The weapons which must not infrequently be enjoined upon the radiologist are posturing, manipulations and perseverance.
  9. When carcinoma (or other lesion) of the gastro-intestinal tract is a possibility and the radiological findings are negative, the clinician must see to it that his patient has had efficient radiological examination. He must critically scrutinize the skiagrams. If these are defective in some respect it can be assumed that the fluoroscopy also was defective.
  10. When the clinician, suspecting malignant disease, is satisfied that the possibilities of radiology have been exhausted for the time being, he must either advise laparotomy or set a strict time limit—let us say six weeks—beyond which the indefinite continuation of medical treatment is unjustifiable. At the end of this period the patient should be reexamined clinically and radiologically and the position reviewed. Delay is more dangerous than diagnostic laparotomy. The former has a considerable mortality. The latter should have practically none.
- I should like to point out that although the patients referred to in this article have eventually come under my care, their previous investigations were carried out by many different clinicians and radiologists in private practice and in several public hospitals, not necessarily in that one to which they finally applied for help.

#### Acknowledgements.

In conclusion, I should like to express my indebtedness to Sir H. B. Devine for teaching me the spirit of attack in diagnosis and the control by the clinician of gastro-intestinal radiology.

#### Legends to Illustrations.

FIGURE I.—Hiatus hernia and ulcer of the duodenum. The hernia is usually not mentioned in the radiologist's report.

FIGURE II.—Case V. Mr. M. The stomach was reported as normal, but it has a huge lesser curvature ulcer, which fails to retain barium in the usual upright position. Its presence is suggested by the radiating rugæ. Its upper limit is marked by a small crescent of air, its lower limit by a small crescent of barium.

FIGURE III.—Case VII. Mrs. R. Unreported carcinoma of the lesser curvature.

FIGURE IV.—Case VIII. Mr. A. Pyloric obstruction with unreported massive carcinoma of the lesser curvature.

FIGURE V.—Case VIII. Mr. A. Eighteen months later. A gastro-enterostomy has relieved the obstruction. The growth starting on the lesser curvature now extensively involves the greater curvature at the pylorus.

FIGURE VI.—Case IX. Mr. V.D.A. First examination. An unreported carcinoma high on the lesser curvature.

FIGURE VII.—Case IX. Mr. V.D.A. Second examination, twenty-two months later. The carcinoma still unreported. It has spread from the upper to the lower arrow. The velvety outline of the growth now reaches the angular deformity at the lower arrow, and can be appreciated by comparing the edge of the lesser curvature shadow on either side of the lowest arrow. A lens helps.

FIGURE VIII.—Case IX. Mr. V.D.A. Third examination, five months later. Carcinoma still unreported. The angular deformity and the irregularity of the lesser curvature are still seen.

FIGURE IX.—Case IX. Mr. V.D.A. Fourth examination, eleven weeks later. The carcinoma can be seen steadily eroding the lesser curvature. It is now reported as "a small penetrating ulcer on the lesser curvature".

FIGURES X, XI and XII.—Case IX. Mr. V.D.A. Fifth examination. A clinician suggested that examinations which used only the upright posture were inadequate. For the first time this patient was now examined in the horizontal position. Note the supine position (Figure XII). The gas is at the pylorus. The still flexible normal wall at the upper pole is ballooned out, the deformity thus being made still more obvious. In fact the growth can be seen to surround the stomach completely. The radiologist reported "the possibility of carcinoma cannot be overlooked".

FIGURES XIII and XIV.—Case IX. Mr. V.D.A. Sixth examination. The growth is now palpable, as large as a fetal head, below the left costal margin. The radiologist reports "a large carcinoma".

FIGURE XV.—Case XI. Mrs. L.W. First examination. Radiologist diagnoses pyloric stenosis.

FIGURE XVI.—Case XI. Mrs. L.W. Second examination; special efforts now show that pyloro-duodenal region is normal.

FIGURE XVII.—Case XI. Mrs. L.W. Third examination. Further special efforts now show the obstruction, a carcinoma, at the duodeno-jejunal flexure.

FIGURE XVIII.—Case XII. Mr. R. McG. Shows a large carcinoma on the lesser curvature, which was not seen by a radiologist using fluoroscopy only.

#### MEDIASTINAL EMPHYSEMA.<sup>1</sup>

By V. L. COLLINS, M.D. (Melbourne), M.R.C.P.,  
D.C.H.,

Honorary Physician to Out-Patients, Children's  
Hospital, Melbourne.

THE escape of air from the normal respiratory pathways into tissues in which it is not normally present can produce a condition which may frequently become serious. Mediastinal emphysema may be a complication of a variety of conditions, and the term spontaneous mediastinal emphysema has been used to describe the state in which air is present in the mediastinum when no underlying disease has been apparent. Though mediastinal emphysema may be caused by trauma and by thoracic operations, it is now recognized that it most commonly follows the rupture of pulmonary alveoli into the vascular sheaths of the lung—the consequent pulmonary interstitial emphysema leads to mediastinal emphysema. The air may remain locked in the mediastinum or may pass into the neck, the pleural cavity or the retroperitoneal tissues. Thus subcutaneous emphysema beginning in the neck,

<sup>1</sup> Read at a meeting of the Melbourne Paediatric Society on June 11, 1947.

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ILLUSTRATIONS TO THE ARTICLE BY DR. V. J. KINSELLA.



FIGURE I.



FIGURE II.



FIGURE III.

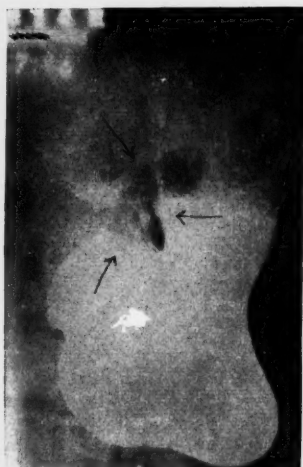


FIGURE IV.



FIGURE V.

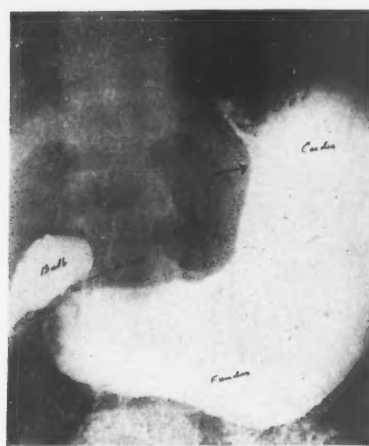


FIGURE VI.



FIGURE VII.



FIGURE VIII.



FIGURE IX.

ILLUSTRATIONS TO THE ARTICLE BY DR. V. J. KINSELLA.

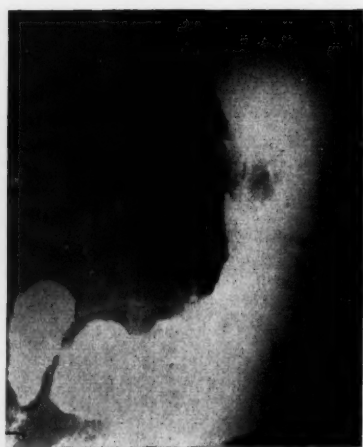


FIGURE X.



FIGURE XI.



FIGURE XII.



FIGURE XIII.



FIGURE XIV.



FIGURE XV.



FIGURE XVI.



FIGURE XVII.



FIGURE XVIII.

ILLUSTRATIONS TO THE ARTICLE BY DR. V. L. COLLINS.

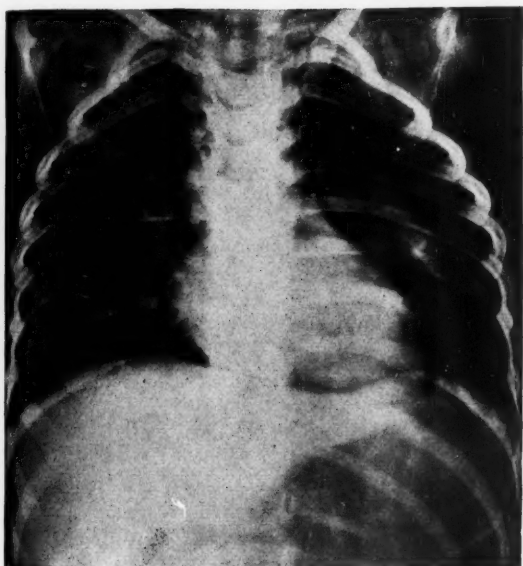


FIGURE I.

Skiagram showing mediastinal and subcutaneous emphysema which occurred in a child, aged three years, suffering from mild bronchitis.

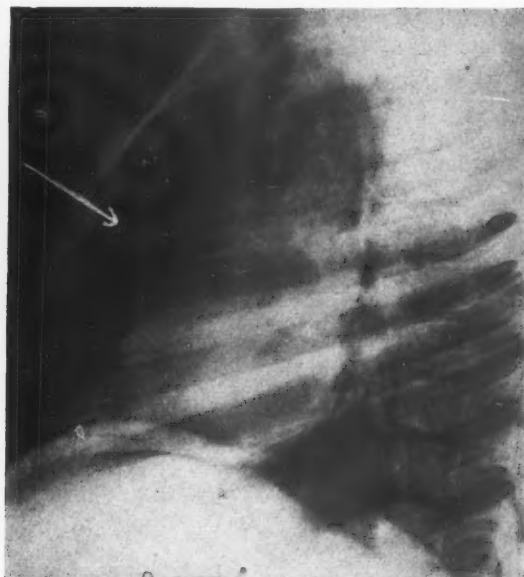


FIGURE II.

Same child as in Figure I; lateral view.

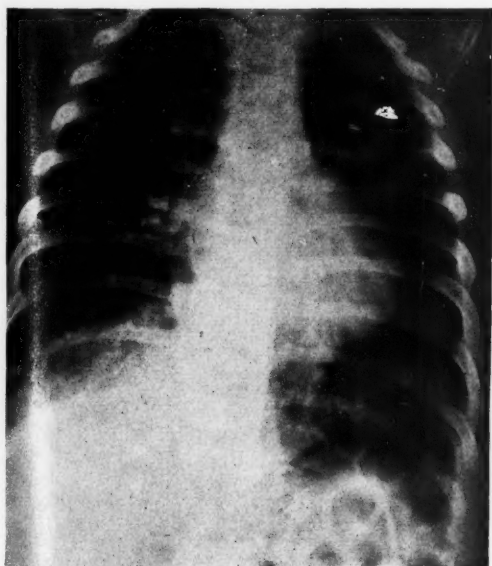


FIGURE III.

Skiagram showing mediastinal emphysema, which occurred in an infant, aged three months, suffering from bronchopneumonia.

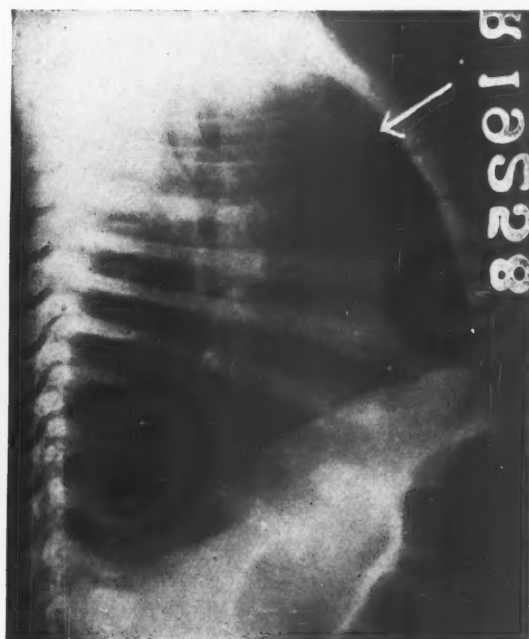


FIGURE IV.

Same infant as in Figure III; lateral view.

ILLUSTRATIONS TO THE ARTICLE BY DR. T. E. LOWE AND DR. E. W. BATE.

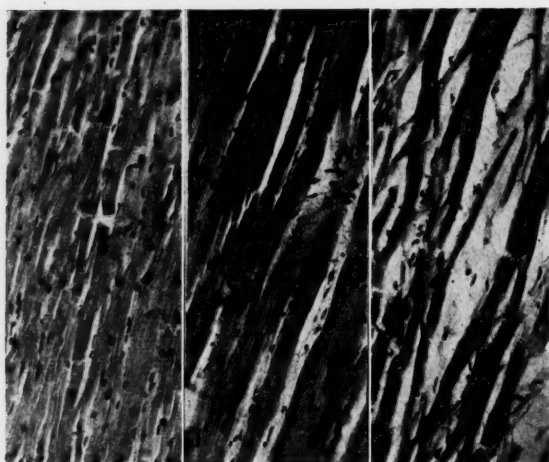


FIGURE I.

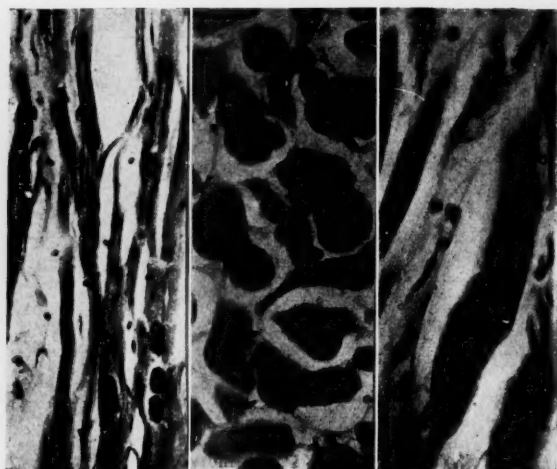


FIGURE II.

ILLUSTRATIONS TO THE ARTICLE BY DR. C. J. OFFICER BROWN AND DR. HAMLEY WILSON.

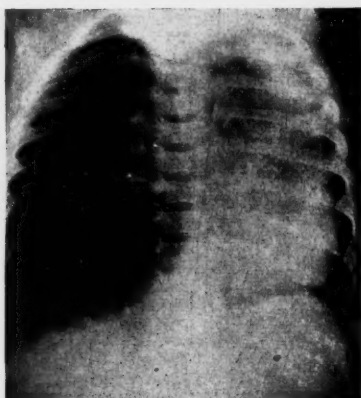


FIGURE I.



FIGURE II.

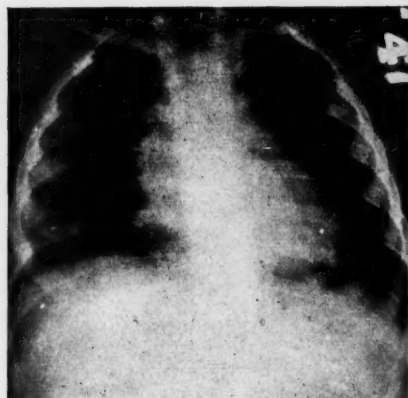


FIGURE III.

ILLUSTRATIONS TO THE ARTICLE BY DR. B. SHORT AND DR. E. H. DERRICK.



Figures showing scabies with gross parakeratosis.





pneumothorax or retroperitoneal emphysema may be an accompaniment of mediastinal emphysema.

Many physicians have observed the presence of subcutaneous emphysema in pulmonary disease; but it was not until Macklin<sup>(1)</sup> demonstrated the series of events which produced this state that its true significance was appreciated. In the influenza epidemic of 1918-1919 many patients were reported with this complication, and some very accurate observations on the changes in these patients were recorded;<sup>(2)(3)</sup> further mention is made of this below. Most of the literature upon the subject of mediastinal emphysema has appeared in the last ten years, and the condition has usually been regarded as benign. Macklin and Macklin, however, in a recent and extremely thorough review of the literature,<sup>(4)</sup> stress the fact that the condition can be frequently serious and at times fatal. The term airblock has been used to describe the state of pulmonary interstitial emphysema and mediastinal emphysema, and there is little doubt that when it complicates a serious lung condition or appears in the newborn, it may be an important factor leading to a fatal outcome.<sup>(5)</sup> Once one knows the series of events which lead to subcutaneous emphysema, it is easy to assume or diagnose the presence of mediastinal emphysema. In the absence of subcutaneous emphysema, however, the diagnosis of mediastinal emphysema will be made only if the clinical features are familiar and if its possibility is kept in mind. It is because of this and because it is considered that mediastinal emphysema is more common and more serious than is recognized that a brief review of the subject has been undertaken.

My interest was first aroused in this subject when I was seeking an explanation for subcutaneous emphysema of the neck and axillæ in a young man suffering from spontaneous hæmopneumothorax and admitted to the North Middlesex County Hospital, London, in 1943. Subsequently a second patient with subcutaneous emphysema came under my care at the same hospital. She was a girl, aged six years, who had been subject to eczema and attacks of bronchitis with spasm since infancy, and she was admitted in a semi-conscious, cyanosed, dyspnoic state. She was wheezing and presented features of bronchopneumonia, with subcutaneous emphysema of the neck, the upper half of the chest and the axillæ. She improved steadily with treatment for her asthma and bronchopneumonia (adrenaline, atropine, oxygen and sulphamezathine), and within three days she was relatively well again.

The history of the following patient is presented in more detail.

#### Report of a Case.

A girl, aged three years, was admitted to the Children's Hospital, Melbourne, at 11.30 p.m. on January 20, 1947. Except for rather frequent upper respiratory infections, she had been well until four days previously, when she awoke with a husky voice. This persisted for two days without any other symptoms; then she developed coryzal features and a cough, but was otherwise well. On the morning of her admission to hospital she was listless and "grizzly", and at 11 a.m. vomited her breakfast. She refused her midday meal, but otherwise appeared bright and seemed well. At 3.30 p.m., after eating a little bread and butter, she was put to bed, and it was then noticed that she was dyspnoic, though no wheezing was apparent. The dyspnoea gradually became worse over the next few hours, and though she was sleeping intermittently, she became restless and apprehensive.

On her admission to hospital at 11.30 p.m. she was dyspnoic, pale, exhausted and apprehensive, and her lips were a little cyanosed. Her temperature was 99° F., her pulse rate was 130 per minute and her respirations numbered 40 per minute. Some expiratory wheezing was heard, resonance was detected over the sternum and the precordial area, and the heart sounds were distant. No abnormal heart sounds were heard. Respiratory excursion was moderate, the breath sounds in the axillæ were diminished and expiratory rhonchi over both lungs and a few basal rales were heard. The abdomen was normal. Subcutaneous emphysema was pronounced over the anterior part of the neck, extending upwards to the angle of the jaw. Her condition improved after an injection of one one-hundred-and-fiftieth of a grain of atropine, and she slept moderately well after a dose of chloral hydrate and sodium bromide.

Radiological examination of the chest revealed, in the antero-posterior films, relatively normal lung fields, a little air in the superior mediastinum and lateral displacement of the broncho-vascular markings in the lower lobes of both lungs. The lateral view revealed a large collection of air in the anterior part of the mediastinum and some air in the posterior part of the mediastinum. The interstitial and subcutaneous presence of air in the neck and axillæ was confirmed. (See Figures I and II.)

On the next day she was still dyspnoic, the subcutaneous emphysema had extended over the upper half of the chest wall, and scattered rhonchi were still present. Thereafter her condition steadily improved, and within four days the subcutaneous emphysema had cleared, and the lungs appeared normal. She was discharged home on February 25, and the X-ray appearances of her chest were normal one week later.

#### Ætiology and Pathology.

Two main questions arise in considering the patients with this condition: how does air escape from the lungs to the subcutaneous tissues and in what circumstances may this occur?

In 1919 Berkley and Coffen<sup>(6)</sup> found eleven patients with subcutaneous emphysema in 1701 cases of bronchopneumonia. They observed that there were "emphysematous excavations" in the lungs, from which air could be traced along the course of the blood vessels to the hilum of the lung. Their assumption that this process commenced with the rupture of air sacs into the vascular sheaths has subsequently been proven by Macklin.<sup>(7)</sup> He found that when air was insufflated through a catheter introduced into the lower lobe of the right lung of an anesthetized cat, the animal died with a double pneumothorax. Examination of the lungs of animals that died in this manner showed that in the insufflated lobes the perivascular sheaths, particularly those of the arteries, were dilated with air. These changes were found to be present from the peripheral small vessels to the hilum. In contrast to this, the interstitial tissue surrounding the bronchial tree contained no air unless a pulmonary artery or vein was adjacent. The final proof of leakage from pulmonary alveoli was a difficult matter. The openings were undoubtedly small, probably closed after the air had passed through, and so were difficult or impossible to see in stained sections, and the passage of a variety of dyes did not help. Subsequently, when fine carmine granules in hot gelatin were introduced into the insufflated portion of the lung, the gelatin was found in the perivascular sheaths and an accumulation of carmine granules at the sites of rupture.<sup>(8)</sup>

Macklin was able to demonstrate conclusively that the pneumothorax was caused by ruptures in the mediastinal pleura permitting the escape of air from the mediastinum to the pleural cavity. These ruptures were usually situated in the posterior mediastinal wall at varying levels, but most commonly just below the hilar region. He found no evidence that air went through the pleura directly.

The production of pulmonary interstitial emphysema from alveolar rupture, therefore, appears the first and essential step in a sequence of events which may lead to mediastinal emphysema, pneumothorax, subcutaneous emphysema and retroperitoneal emphysema. Therefore a search for the clinical causes of pulmonary interstitial emphysema is first necessary. Macklin's work indicates that pulmonary interstitial emphysema may occur in the following circumstances: (a) when pulmonary alveoli are over-inflated, and particularly when this condition is accompanied by increased intraalveolar pressure; (b) when the calibre of the pulmonary blood vessels is reduced, particularly if this reduction is accompanied by increased intraalveolar pressure. Compensatory over-inflation (without increased intraalveolar pressure) commonly occurs in atelectasis, and so may appear in the presence of an obstructed bronchus, a foreign body, pneumonia and atelectasis of the newborn. In many newborn babies with pulmonary interstitial emphysema and its sequelæ, the presence of atelectasis has been demonstrated. The reduction of the calibre of pulmonary vessels alone (as in heart failure, pulmonary embolism or pulmonary stenosis) would appear to be a rare cause of the condition. In forced expiration, however, the combination of increased intra-

alveolar pressure with a reduced amount of blood in the pulmonary circulation and so a decrease in the calibre of the pulmonary vessel, provides both predisposing factors to alveolar rupture.

In the search for an explanation of the rupture of alveoli and the passage of air into the vascular sheaths, a consideration of lung anatomy is helpful. The alveoli may be divided into two types, (a) those which have their bases lying between other alveoli, and (b) those whose bases rest against some structure other than adjoining alveoli. In the latter class the bases will abut upon bronchi, bronchioles, blood vessels, connective tissue septa or pleura. The space between the alveolar bases and the bronchial or vessel lumen is filled by a connective tissue sheath, and normally the volume of this sheath is constant throughout the respiratory cycle. If the alveoli are over-distended with air, nothing will happen to the alveoli whose bases rest upon a bronchus or bronchiole or upon other alveoli, because the same excess quantity of air which distends the alveoli is distending the rest of the airway. But what of the alveoli bordering upon blood vessels? Normally the alveoli are hyperexpanded when the depth of respiration is greater than usual. Under these conditions the amount of blood in the pulmonary arteries and veins is correspondingly increased. However, if the heart cannot keep pace by pumping enough blood into the pulmonary arteries to expand the arterial lumen sufficiently, then the alveoli around the vessels are over-expanded, but the vessel lumen is not correspondingly widened and a pressure gradient is created between the alveoli and the sheath. The alveolar bases are trying to expand—that is, to enlarge the outer rim of the vessel sheath—and the sheath does not follow owing to the inadequate expansion of the vessel lumen. Rupture of the bases takes place and air flows into the vascular sheath.<sup>(4)</sup> Such a pressure gradient is created if over-inflation, forced expiration or diminution of the calibre of the vessels is present. However, this cannot be regarded as the only factor concerned with alveolar rupture. If it was so, pulmonary interstitial emphysema and its sequelæ would be an extremely frequent occurrence. It would be expected, for instance, in every woman straining in labour or in every asthmatic patient. Therefore, it seems that some congenital weakness of the alveolar walls must be assumed to account for the occurrence of this condition in only occasional patients subjected to the same physical forces. Toxic factors, such as an influenza infection, also appear to play a part in increasing the possibility of alveolar rupture. In infancy and childhood, too, pulmonary interstitial emphysema seems to occur more readily than in adult life.

The following are the main causes of pulmonary interstitial emphysema and its sequelæ: (i) extraneous causes: trauma—injury to the chest with or without fracture of ribs, operations on the chest including induction of pneumothorax, tracheotomy; (ii) result of factors discussed earlier—atelectasis of the newborn, bronchopneumonia and, lobar pneumonia, asthma, foreign body in a bronchus, insufflation anaesthesia, straining with closed glottis (for example, heavy lifting, childbirth, straining at stool), whooping-cough or any violent cough, attempted resuscitation of the newborn, cardiac disease; (iii) spontaneous rupture of alveoli (possibly).

The first two causes need not enter further into this discussion. There are many reported cases in the newborn and these are considered more fully below. With the knowledge of mediastinal emphysema as a clinical entity, it seems likely that the condition should be recognized more frequently in bronchopneumonia, especially in children. It appears to have occurred particularly frequently in influenza bronchopneumonia but is uncommon in pneumococcal pneumonia. It is not uncommon in bronchial asthma, and Schwartz<sup>(7)</sup> recently analysed 25 cases of subcutaneous emphysema previously reported in the literature. The presence of a foreign body in a bronchus may favour two factors which commonly cause alveolar rupture—over-inflation from atelectasis and increased intraalveolar pressure from coughing to expel the object. Pulmonary interstitial emphysema and its

sequelæ are well known complications of a foreign body in a bronchus;<sup>(4)(8)</sup> but the real cause has often been overlooked when the presence of air is attributed to trauma caused by attempts at removal of the offending object.

Because of the not infrequent appearance of subcutaneous emphysema following operations, it has been called "surgical emphysema". Macklin states<sup>(4)</sup> that it appears to be much more common after those operations in which the anaesthetic has been given intratracheally than in those in which it has been given by the inhalation method. Sometimes it may arise because atelectasis has occurred after operation, but in other cases the pressure of the anaesthetic agent entering the lungs is the obvious cause. In whooping-cough, subcutaneous emphysema was found once in 1200 cases;<sup>(4)</sup> but the frequency of pulmonary interstitial emphysema was probably much greater. Hamman<sup>(9)(10)</sup> has recorded the appearance of spontaneous mediastinal emphysema in apparently fit persons with no evidence of pulmonary disease. Macklin, however, has pointed out that more detail would be needed to exclude the usual factors—for example, the possibility of recent straining or of a recent respiratory infection.

In any one of these conditions the following sequence of events may occur. Air passes through multiple and minute ruptures of the alveolar walls to enter the perivascular sheaths and then progresses along the blood vessels to reach the mediastinum. The air may remain locked in the mediastinum or may extend in four directions—upwards to the neck producing subcutaneous emphysema which usually appears at the jugular notch, to the pleural cavity on one or both sides, to the interstitial tissues of the opposite lung, or downwards to the retroperitoneal tissues. Occasionally it ruptures into the pericardial sac, and sometimes pneumoperitoneum is produced. Often peripheral extension of pulmonary interstitial emphysema occurs, with the formation of subpleural blebs of varying size. Macklin considered that these subpleural blebs rarely ruptured to produce pneumothorax, and that the usual mode of production of pneumothorax was a rupture of the mediastinal pleura following an increase in tension there. Certainly this mechanism would appear the most probable in the production of bilateral pneumothorax, and pneumothorax has been observed clinically to follow mediastinal emphysema.<sup>(11)</sup> However, in a recent report<sup>(12)</sup> on six newborn babies suffering from pneumothorax, it has been shown that though all had pulmonary interstitial emphysema and subpleural blebs, only one had mediastinal emphysema. The authors concluded that pulmonary interstitial air could rupture subpleural vesicles in the newborn. Macklin had observed this happen experimentally in two of three rabbits; but both he and Hamman consider that bilateral pneumothorax and tension pneumothorax usually result from rupture of the mediastinal wall and that the same factor is a more common cause of spontaneous pneumothorax than is generally assumed. Subcutaneous emphysema may at times affect the whole body and extend from fingers to toes.

From what has been said, it will be realized that from a variety of causes the sequelæ of pulmonary interstitial emphysema may result in a diverse clinical picture. Pulmonary interstitial emphysema alone or with mediastinal emphysema may be present. When the interstitial emphysema is extensive, it has a splinting effect on the lungs, for the air being trapped in the lungs holds it in a state of inflation and prevents deflation. This effect on respiratory excursion, together with interference with the pulmonary circulation occasioned by compression of the vessels by perivascular collections of air, may have a serious outcome, especially if it is complicating other pulmonary disease. If in addition pneumomediastinum is present, the outcome may be fatal. It is difficult to understand why, in some cases, the air becomes trapped in the mediastinum while in others it readily extends to the pleural cavity or to the subcutaneous tissues. If the mediastinal pressure is not relieved by natural process or otherwise, pressure on the great vessels occurs. The term airblock<sup>(4)(13)</sup> has justifiably been applied to the condition, and removal of air from the mediastinum may relieve alarming symptoms.<sup>(13)</sup>

### Clinical Picture.

The clinical features of pneumothorax are familiar to all. It is important to stress the clinical picture produced by air in the mediastinum and pulmonary interstitial tissues when such aids to diagnosis as subcutaneous emphysema and pneumothorax are absent.

The outstanding clinical features in a patient with considerable mediastinal emphysema are dyspnoea and cyanosis. There is poor respiratory excursion in a chest held in a position near full inspiration, and cardiac dullness is obliterated. Peculiar heart sounds, distant heart sounds and engorged neck veins may be present, and the radiological appearance is typical. Subcutaneous emphysema may be the only external sign of mediastinal emphysema and pneumothorax may be present. In an infant the presence of dyspnoea and cyanosis together with fixation of the chest in a position of full inspiration should immediately suggest mediastinal emphysema.

Torrey and Grosh<sup>(6)</sup> gave an excellent description of the condition in 1919, when describing a severe outbreak of influenzal bronchopneumonia in a military camp. They considered that the majority of the 1800 patients had some degree of what they called "acute pulmonary emphysema". They describe the course of the disease as follows:

The point that struck us here with great force was the intense dyspnoea, with little cardiac disturbance, cyanosis, great air hunger, and erection of the chest fixed in a state of hyperinspiration with only tidal air movement. As the muscles of respiration failed *exitus* occurred, a respiratory death in contradistinction to the toxic, circulatory or vasomotor death commonly seen in the early days of true pneumonia. The pulse in desperately ill patients was excellent in volume and quality, and relatively slow. As this distension of the chest developed it could be noted that the whole chest excursion was diminishing as manifested by a general diminution of the lateral excursion of the costal arches until there was an inspiratory retraction, more in the affected side at the onset, then becoming bilateral.

Very soon the accessory muscles of respiration were the only effective ones giving a lift of the upper chest, while over the rest of the thorax was seen only a weak effort of the muscles, giving an undulatory movement of the chest already so overdistended that any gain in capacity was impossible. Sternal tympany became more marked, assuming a box-like character. Marked stasis of all the veins entering the chest and those drained by the superior *vena cava* was manifest, cyanosis became more profound and in the end stage there was an ashen lividity of the whole face and chest.

When in apparent respiratory extremis, frequently a patient would begin to complain of pains, substernal and in the jugular fossa, and crepitation would be noted in the subcutaneous tissue at the root of the neck, and immediate and marked subjective relief was apparent, rapidly followed by a noticeable improvement in the respiratory excursion of the chest, and the most striking decrease in cyanosis and jugular distension. With the relief of cerebral venous stasis a clearing up of the previously accumulative mental hebetude occurred with remarkable suddenness. Cutaneous emphysema soon became diffuse, not infrequently spreading down the trunk to the pubes, first appearing along the sheaths of the cutaneous veins. As the intrathoracic pressure was thus relieved these apparently moribund patients often went on to recovery.

Though there is usually dramatic relief of the symptoms with the appearance of subcutaneous emphysema (and sometimes with pneumothorax), death can occur even in the presence of subcutaneous emphysema,<sup>(6)</sup> or surgical intervention may be required.<sup>(6)</sup>

In adults pain is a common symptom.<sup>(6)</sup> Pain may appear suddenly over the front of the chest, in the shoulders or down the arms, and such pains, together with dyspnoea, can produce a clinical picture simulating coronary occlusion. Occasionally abdominal pain caused by the presence of retroperitoneal emphysema may be the prominent early symptom. Hamman, who appears to have been the first to recognize mediastinal emphysema as a clinical entity,<sup>(9)</sup> diagnosed this condition in his first patient only in retrospect, but in all reported seven patients suffering from spontaneous mediastinal emphysema in four years.<sup>(9)(10)</sup> He emphasized the peculiar heart

sounds which may occur. These are usually a crunching, crackling or bubbling type of sound, which occurs over the precordium with each systole, and which occasionally may be heard several feet from the patient. They are pathognomonic of that type of mediastinal emphysema in which the air has worked around to the front of the pericardial sac. Hamman's observations were made mainly in adults, but in children these sounds are rarely recorded. In infants particularly the most important physical signs are the obliteration of cardiac dullness with a hyperresonant percussion note over the sternum and distant heart sounds.

The radiological findings are usually typical and definitely establish the diagnosis. There appears no doubt that in the past mediastinal emphysema has frequently been overlooked because it produced so few changes in an antero-posterior film and the taking of a lateral picture has been omitted. The antero-posterior view may reveal a linear shadow of air along either side of the superior part of the mediastinum or along the borders of the heart; but in the case of the newborn particularly these views may reveal no evidence of mediastinal emphysema. The lateral film offers the most conclusive evidence and in most instances will prove to be diagnostic. In this view one characteristically sees air just beneath the sternum, and if there is a large collection the heart will be displaced posteriorly. Air may also be seen in the posterior as well as in the anterior part of the mediastinum. The lateral view, however, may be misleading in the presence of pneumothorax,<sup>(11)</sup> particularly a tension pneumothorax. In such circumstances the lateral film may reveal the presence of substernal air in the absence of mediastinal emphysema, probably because of the herniation of the anterior part of the mediastinum.

### Spontaneous Mediastinal Emphysema and Pneumothorax in the Newborn.

As both spontaneous mediastinal emphysema and pneumothorax are usually a sequel of pulmonary interstitial emphysema in the newborn, it is reasonable to consider them together. Subcutaneous emphysema has been reported on a number of occasions in the newborn but has been considered one of the rarities of medicine. Pneumothorax, however, is not uncommon. Davis and Stevens<sup>(12)</sup> in 1930 made a routine radiographic examination of 702 newborn infants and found pneumothorax in six of them—that is, in approximately 1% of babies on the third day of life. In 1940 DeCosta<sup>(13)</sup> found in the world literature 67 instances of pneumothorax occurring during the first few months of life. Of these he reviewed 46 instances which occurred in the first few days of life and reported two further cases of his own. From this review he concluded that there were two distinct forms of pneumothorax, which he called *pneumothorax abrupta* and *pneumothorax lenta*. In the former, smaller group the symptoms dated from birth or shortly thereafter, the baby was deeply cyanosed, dyspnoea was pronounced and stridor was sometimes present; there were signs of pneumothorax and the mortality rate was 42%. In the latter group symptoms were mild and often included gradual onset of cyanosis, perhaps noticed only when the baby cried, rapid respirations were present, and the physical signs of pneumothorax were more difficult to recognize. Recognition of this type of pneumothorax was frequently late and often the discovery was accidental, and none of this group died from the pulmonary condition. Cardiac anomalies were found four times in the series of 69 patients—a finding which agrees with the suggestion made above concerning the manner in which congenital heart disease may favour the production of pulmonary interstitial emphysema.

Salmon, Forbes and Davenport have recently reported six newborn infants dying from airblock.<sup>(14)</sup> All had pneumothorax and pulmonary interstitial emphysema, and one had also mediastinal emphysema. They emphasized that in the presence of pulmonary interstitial emphysema an apparently small or moderate pneumothorax might be under very high tension. This can be readily appreciated, for the air trapped in the lung tissue prevents more than moderate collapse.

A number of newborn infants have been reported with features of mediastinal emphysema only. Gumbiner and Cutler<sup>(14)</sup> report the recovery of a newborn baby after aspiration of air from the mediastinum. The infant suddenly became dyspnoeic and cyanosed thirty hours after birth. There was no subcutaneous emphysema or bulging of the precordium, but radiological examination confirmed the diagnosis. The infant's condition became progressively worse after the next twenty-four hours and so a needle attached to a 20-millilitre syringe was introduced into the third left intercostal space and directed medially parallel with the inferior surface of the sternum. They record that when this was done "there was sudden expulsion of the plunger of the syringe to the 6 c.c. mark and within five minutes there was a dramatic change in the baby's condition". Within three days the infant was clinically well. After appreciating the value of the lateral X-ray film in the diagnosis of mediastinal emphysema, these authors recognized three additional cases during the next four months. Successive radiological examinations of one of these patients were of interest, as they revealed an increasing mediastinal accumulation of air and finally spontaneous pneumothorax coincident with clinical improvement and evidence of reduction in mediastinal emphysema.

Smith and Bowser<sup>(15)</sup> report two babies with mediastinal emphysema, both of whom were cyanosed at birth. Both recovered, one after the introduction of a needle under the sternum in the fourth right intercostal space permitting the escape of air under pressure, and the other without any attempt at removal of mediastinal air. Fisher<sup>(6)</sup> mentions a baby, normal at birth, who was noticed three and a half hours after birth to be cyanosed and to have blood-tinged froth exuding from the mouth, and who died within six hours after birth. Autopsy revealed mediastinal emphysema with bilateral pulmonary interstitial emphysema and atelectasis of part of the left lung.

#### Treatment.

It is probable that in most cases mediastinal emphysema, whether in infants or children, is slight and causes no symptoms. In others the symptoms are mild and spontaneous recovery will probably ensue. Symptomatic treatment with perhaps the administration of oxygen is all that will be required. However, if the mediastinal pressure rises too high, or if air escapes to both pleural cavities causing bilateral pneumothorax, or if a serious inflammatory condition already exists in the lung, then vigorous and urgent therapeutic measures may be indicated. If a large pneumothorax or tension pneumothorax is present, aspiration of air will be necessary. If a tension pneumothorax recurs it may be advisable to perform a closed thoracotomy and allow air to escape under water. If the air is locked in the mediastinum, removal of the air from the anterior part of the mediastinum as described above may be a life-saving measure. Repeated aspiration may be necessary, or occasions may arise when it is advisable to leave a needle or cannula in the anterior part of the mediastinum connected by tubing to a water trap. More vigorous surgical measures, such as cervical mediastinotomy, may be required,<sup>(6)</sup> and splitting of the sternum has been recorded.<sup>(10)</sup>

#### Summary and Conclusions.

1. The clinical features, aetiology, pathology and treatment of mediastinal emphysema are described.
2. Mediastinal emphysema may arise from a variety of causes which produce alveolar rupture with the passage of air into the perivascular sheaths, causing pulmonary interstitial emphysema and extending to the mediastinum. Its relation to spontaneous pneumothorax is discussed.
3. The main clinical features are dyspnoea, varying degrees of cyanosis, almost complete fixation of the chest near the inspiratory position, obliteration of cardiac dullness, distant and peculiar heart sounds, subcutaneous emphysema and signs of pneumothorax. The diagnosis is established with the aid of a radiological examination, and the pathognomonic appearance of the lateral view is stressed.

4. The history of a patient suffering from mediastinal emphysema with subcutaneous emphysema is recorded.

5. A brief review of spontaneous mediastinal emphysema and of pneumothorax in the newborn is made. It is considered that the condition is more common in the newborn and in infants suffering from bronchopneumonia than is commonly recognized.

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#### Addendum.

Since the above-mentioned patient was presented, two further patients with this condition have been seen at the Children's Hospital. One had a foreign body in a bronchus causing mediastinal and subcutaneous emphysema and pneumothorax; the other, a baby of three months, had bronchopneumonia and mediastinal emphysema with severe symptoms dramatically relieved by repeated substernal needling (see Figures III and IV).

#### HYPERPLASIA OF CARDIAC MUSCLE FIBRES.

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THE occurrence of hyperplasia in mature cardiac muscle has been a much disputed subject for many years. Kölliker (1852), Rokitsky (1856), Goldenberg (1886) and King (1940) amongst others have either stated that it does occur or have accepted the possibility that it can occur. *Per contra*, Mönckeberg (1924), Karsner *et alii*

(1925) and Aschoff (1936) amongst modern writers maintain that enlargement of cardiac muscles occurs only by fibre hypertrophy.

As a result of this lack of agreement between authorities, editorial comment in the *British Heart Journal* (1940) has suggested that direct proof of hyperplasia would be given by (i) the presence of mitotic figures in the muscle nuclei, and (ii) demonstration that the total count of muscle fibres in the heart was increased.

Mitotic figures have been noted in enlarged hearts in infants and children by MacMahon (1937) and in the young adult in muscle repair by King; but apart from these observations the presence of mitoses has rarely been recorded.

Karsner *et alii*, and Dammin and Moore (1939) have attempted to count the total number of muscle fibres in normal and hypertrophied hearts. They have shown a correlation between nuclei numbers and fibre counts, which indicates the absence of multiplication of fibres in the few hypertrophied hearts examined.

In view of this disagreement on so fundamental a problem, it is of importance to record the following observations on a heart in which the authors consider hyperplasia of the muscle fibres to have occurred. Whilst direct proof is not advanced, the indirect evidence in this case is so strong that the presence of hyperplasia must be accepted.

of the three inner layers is skewed towards smaller fibre size.

#### Discussion.

In a previous paper the writers (1948) showed that in simple hypertrophy of the left ventricular muscles the degree of enlargement of the mean fibre diameter was uniform in each layer and that the distribution of the sizes of these enlarged fibres followed a normal curve in each muscle. These observations on simple hypertrophy contrast with this heart, in which the degree of enlargement of fibres in each layer is not uniform, and the distribution of fibre sizes, except in the outer layer, does not follow a normal curve but a curve skewed towards smaller fibre size.

There are two major factors which could lead to enlargement of cardiac muscle fibres—increased mechanical stress and metabolic agents. Of these increased mechanical stress is well recognized as a cause of muscle fibre enlargement, and must have existed in this case because of the aortic valve stenosis. Growth stimuli of metabolic origin are likewise accepted. These might be expected to be effective in rheumatic lesions of the heart, in which tissue destruction and inflammatory responses are present. Both of these factors (increased mechanical stress for physical reasons and metabolic factors because rheumatic lesions are present in all layers) should in the present instance be distributed uniformly throughout the layers.

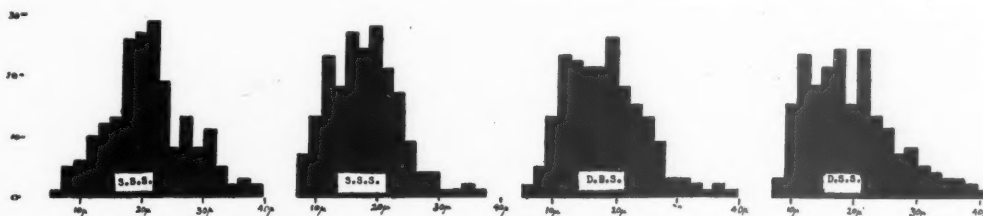


FIGURE III.—Histograms showing frequency distribution of fibre diameters in each muscle.

#### Methods and Material.

The heart examined (number 132) was obtained some years ago at post-mortem examination of a young man, aged twenty-two years, who dropped dead whilst dancing. No clinical history is available.

The heart weighed 82 ounces (2340 grammes) when fresh. Its most notable feature was the enormous left ventricular wall, which was approximately twice as thick as normal, and enlargement of the ventricular cavity. The right ventricle appeared as a small appendage to the left. There was a gross aortic valvulitis with marked stenosis.

Histological examination revealed evidence of syphilis of the aorta and chronic rheumatic myocarditis in all layers of the ventricular muscle. In the three inner layers of the left ventricle many of the cardiac fibres were split longitudinally into two smaller fibres, each with its own nucleus. This splitting of fibres appeared to be quite distinct from the normal branching and anastomosing of cardiac fibres, in which all the fibres concerned are of approximately the same size. There were also many fibres containing two adjacent nuclei (Figures I and II). The absence of any other characteristic features showed that these were not Purkinje fibres.

Measurements of the diameters of the cardiac muscle fibres in the left ventricular wall were made with the technique previously described (Lowe and Bate, 1948). These readings were grouped according to the individual muscles concerned. Only those fibres which were histologically normal were measured.

The mean diameter and standard deviation of fibres in the various muscles are recorded in Table I and the frequency distribution of fibre sizes in the various muscles of the heart is shown by a histogram (Figure III).

These results reveal a lack of uniform enlargement of the mean fibre diameter in the various layers. The histograms show that whilst the frequency distribution of fibre size in the outer layer follows a normal curve, that

The skewness of the distribution of fibre size in the inner three layers could be accounted for by two hypotheses. In the first place it might be assumed that there had been irregular enlargement of the fibres in the layers, so that only some had reached the "limit of hypertrophy" whilst most had enlarged to a lesser degree. This would weight the smaller size end of the histogram; but in view of the enormous increase in the left ventricular muscle mass and the probability that the known stimuli to enlargement are uniformly distributed, this hypothesis

TABLE I.  
Fibre Size in Left Ventricular Wall.<sup>1</sup>

Layer.	Mean Diameter.	Standard Deviation.
S.B.S. (external) . . . . .	21.0μ	±6.58μ
D.S.S. . . . .	19.6μ	±7.4μ
D.B.S. . . . .	18.4μ	±6.15μ
S.S.S. (internal) . . . . .	18.0μ	±5.52μ

<sup>1</sup> S.B.S. = superficial bulbo-spiral muscle; D.B.S. = deep bulbo-spiral muscle; S.S.S. = superficial sino-spiral muscle; D.S.S. = deep sino-spiral muscle.

seems untenable. Alternatively the skewness could be explained by assuming that multiplication of fibres had occurred. This would imply that one large fibre would be replaced by two or more smaller ones. Such an event would distort the histogram in the observed fashion. That the new fibres should be smaller than their parent fibres is to be expected from a consideration of the diffusion of metabolites to and from muscle fibres and capillaries.

The histological appearances seen in these layers—namely, the splitting of muscle fibres and the presence of

multiple nuclei per fibre—are also explicable on the hypothesis of hyperplasia of fibres, but not by that of incomplete hypertrophy.

This discussion leads to the conclusion that whilst the criteria of hyperplasia previously discussed are not satisfied in this instance, the occurrence of hyperplasia is the only adequate explanation of both the histological features and the unusual frequency distribution of the small fibres in the greatly increased bulk of three of the layers of the left ventricular wall.

The difference in behaviour of the outer from the inner layers also provides striking confirmatory evidence for the functional independence of the ventricular muscles, which has been considered in a previous paper (Lowe and Wartman, 1944).

#### Summary.

In this paper are recorded measurements of the diameters of cardiac muscle fibres and their histological appearances in the muscle layers of the left ventricle of an enormous heart. These measurements and the histological appearances of the muscle lead to two conclusions: first, that hyperplasia of cardiac muscle has occurred in some layers, and secondly, that the individual muscles of the ventricles have reacted independently to the disease state.

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#### Legends to Illustrations.

FIGURE I.—Photomicrographs of fibres in D.B.S. muscle from three hearts: (a) normal, (b) simple hypertrophy, (c) hyperplasia. (Magnification the same in each case. Hematoxylin and eosin stain.)

FIGURE II.—Photomicrographs showing multinucleated cardiac muscle fibres. (Hematoxylin and eosin stain.)

### Reports of Cases.

#### GIANT TENSION CYST OF THE LUNG SIMULATING TENSION PNEUMOTHORAX.

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CONGENITAL CYSTS of the lung are not uncommon, and the case here presented illustrates in a striking manner one of the most intriguing clinical syndromes they may present.

#### Clinical Record.

P.C., a female patient, aged eight months, was admitted to the Alfred Hospital on May 11, 1947. The child had been normal at birth, but the mother noticed that she sometimes became breathless during feedings. At the age

of five months she had had an illness which was diagnosed as pneumonia, and after this she had had recurring attacks of severe dyspnoea with some cough and cyanosis. She was admitted to hospital in Hobart for investigation and presented the clinical and radiological signs of a right tension pneumothorax. A needle was passed into the air space and the air found to be under positive pressure. Some 100 millilitres of air were withdrawn and the pressure became negative; but after a short time it was again positive, suggesting that the space communicated with a bronchus by a one-way valve mechanism.

On her admission to the thoracic surgical unit at the Alfred Hospital the child was slightly underweight, but otherwise appeared normal. The trachea was in the mid-line and the apex beat was difficult to palpate. The heart sounds were clear but faint. Movement of the right side of the chest was restricted, the percussion note on this side was hyperresonant, and breath sounds, vocal resonance and vocal fremitus were all diminished. X-ray examination revealed complete collapse of the right lung, with an air space filling the whole of the right side of the chest and extending across the mediastinum almost to the chest wall on the left side (Figure I). The mediastinum and heart were displaced a long way to the left. In the lateral film an oval space in the lower anterior mediastinum showed where the air crossed the mid-line and was an arresting feature of the radiograph (Figure II). Just to the right of the shadow of the collapsed lung in the postero-anterior film there was an elongated vertical shadow resembling a stick of spaghetti, and it was suggested that this might be the edge of a collapsed cyst whose rupture had caused a spontaneous pneumothorax. It seemed more likely that the whole air space was a giant tension cyst, and one of us (H.W.) suggested that this shadow might represent a blood vessel, and this proved to be the true explanation.

Operation was performed on May 27, One hour before operation an injection of morphine ( $\frac{1}{100}$  grain) and atropine ( $\frac{1}{200}$  grain) was given, and 50,000 units of penicillin were injected intramuscularly. Anaesthesia was induced with cyclopropane and oxygen, and a cannula for blood transfusion was inserted in the left arm; 1.5 milligrammes of tubocurarine were injected into the blood transfusion apparatus. An endotracheal tube was passed and anaesthesia was maintained with cyclopropane and oxygen administered by Dr. R. H. Orton with the controlled respiration technique.

With the child in the lateral position the seventh right rib was excised and the pleural cavity was opened. A thin-walled cyst immediately bulged through the wound and had to be deflated by aspiration to allow exposure. The cyst was found to occupy almost the whole of the right pleural cavity and to extend through the anterior mediastinum nearly to the left wall of the chest. It was loosely adherent, but was easily freed, and was found to arise by a small pedicle from the diaphragmatic surface of the medial basal segment of the lower lobe of the right lung. Several large dilated vessels ran through this pedicle and spread out in the wall of the cyst, and it was one of these vessels that produced the spaghetti-like shadow in the pre-operative films. The pedicle was clamped and divided, the cyst was removed, and three mattress sutures of fine silk were inserted to close the pedicle.

The upper and middle lobes of the right lung were completely atelectatic, but the lower lobe, which presented behind the cyst, contained some air. After removal of the cyst the lower lobe rapidly became fully expanded, but the upper and middle lobes were still dark and only partially aerated when the chest was closed. The chest wall was closed in layers with interrupted sutures of fine silk, and 100,000 units of penicillin were left in the pleural cavity after all air and fluid had been withdrawn.

Convalescence was uneventful. On the second day the child was taking all her food; she was allowed out of bed on the third day and was discharged from hospital, completely well, on the fourteenth day. X-ray examination on the seventh day after operation revealed completely normal lung fields with the mediastinum in the middle line (Figure III).

### Comment.

This condition should always be suspected in young infants suffering from recurrent attacks of dyspnoea or cyanosis, and of course all these children should be radiologically examined. It is impossible to differentiate in some cases between a tension cyst and a pneumothorax; but because spontaneous pneumothorax is extremely rare in young infants, the diagnosis of tension cyst should be made in most cases.

The only effective form of treatment for these patients is excision of the offending cyst, and in some cases it may replace most of the lung and pneumonectomy may be required. In an emergency, aspiration of air or the insertion of a water seal closed drain may give temporary relief; but it should be recognized that however desperate the child's condition may appear, radical surgery at the earliest possible moment gives it its greatest chance of survival.

### Legends to Illustrations.

FIGURE I.—Postero-anterior film of the chest before operation. The mediastinum is displaced to the left and the cyst can be seen extending almost to the left chest wall. The spaghetti-like shadow to the right of the collapsed right lung was found at operation to be a large blood vessel in the wall of the cyst.

FIGURE II.—Right lateral film of the chest before operation. The large oval window where the cyst traversed the anterior mediastinum is well shown.

FIGURE III.—Postero-anterior film of the chest seven days after operation, showing normal appearance of the heart and lung.

### A REMARKABLE CASE OF SCABIES.

By B. SHORT,

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and

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IN 1946 an aboriginal boy, aged about seventeen years, came under observation at Palm Island because of an extraordinary condition of the skin. Large, white, friable excrescences adorned his head, neck, trunk and all four limbs (see illustrations). They were itchy. He was always scratching and slept badly. The disease affected also the finger-nail beds; the nails themselves were normal, but the tips were lifted away from the fingers by the subungual lesions.

The condition had been present for about six years and had failed to respond to starch poultices, salicylic acid preparations, and the intravenous and intramuscular administration of arsenicals, of which he had had about 100 injections. Material was scraped off the lesions and submitted for investigation.

Microscopic examination showed the skin scrapings to consist of greatly hypertrophied, but still nucleated, *stratum corneum*. It was arranged in a loose network, and the intervening spaces were, to our astonishment, inhabited by numerous *Sarcoptes scabiei*. No fungus could be seen in the sections or grown on attempted culture.

He was treated with both sulphur ointment and an emulsion of benzyl benzoate after the use of starch poultices and salicylic acid to soften the keratin. Treatment had to be energetic and sustained, for the condition tended to relapse. However, after some months his skin condition became normal except for one residual patch on the right buttock. He put on about two stone in weight and became a new man.

Reference to the literature showed that cases of long standing, untreated scabies accompanied by much "crusting" were recorded under the name "Norwegian scabies". They had been observed particularly among lepers in Norway. No convincing explanation was offered why certain patients should react with gross parakeratosis. Debilitation by insanitary living conditions or by diseases such as leprosy or tuberculosis was suggested. Our patient had no sign of leprosy or tuberculosis. Apart from the

misery induced by the continual pruritus and the scorn of his fellows, he was in good health.

By a curious coincidence a specimen of skin had been received in Brisbane from the same patient five years earlier, when he lived in north-west Queensland, and the same diagnosis had been made. He apparently had escaped specific treatment on that occasion.

Scabies is common among the aborigines and half-castes at Palm Island. Two other patients observed recently have presented an appearance somewhat similar to that of the patient here described; they responded to sulphur treatment. Another atypical feature is the distribution of the lesions in babies, sites of predilection being wrists, ankles and scalp. Some of the parents dig the mites out with a needle. Scabies has caused the death of several infants from septicæmia following secondary infection.

### A CASE OF PRIMARY MENINGOCOCCAL CONJUNCTIVITIS.

By NANCY LEWIS, M.D., D.O., F.R.A.C.S. (Oph.),  
and

A. A. FERRIS, M.B., B.S.,  
*Melbourne.*

THE meningococcus (*Neisseria meningitidis*) is not a common ætiological agent in the development of ocular infection and may occur in the conjunctiva with or without pathological symptoms.<sup>(1)</sup>

Conjunctivitis due to the meningococcus, unlike that caused by the *Neisseria gonorrhæa*, is a relatively mild condition. It is usually unilateral, either catarrhal or purulent in character, and may be associated with corneal ulceration and enlargement of the preauricular gland. It most frequently occurs in association with cerebro-spinal fever, and, prior to the use of antimeningococcal serum in treatment, it was not an infrequent accompaniment of this disease. In such cases it occurs early and may be of diagnostic importance.<sup>(2)</sup> Reece has reported a case of cerebro-spinal meningitis in which the conjunctiva may have been the portal of entry of the meningococcus, but his evidence is unconvincing.

Acute meningococcal conjunctivitis without any signs of meningitis has been considered rare, very few cases having been reported in the literature until 1944. A bibliography is given by Mangiaracine and Pollen,<sup>(3)</sup> who themselves observed 10 cases within a five-month period. Male children between the ages of fourteen weeks and fifteen years were affected. In two patients the condition was a manifestation of a meningococcal septicæmia, while in eight patients it was a purely conjunctival disease. Of the latter, four were due to a Group I meningococcus, two to a Group II strain, and two were untyped. The eye condition rapidly responded to sulphonamides administered locally and orally, and in one case responded to zinc salts. There was no residual ocular change.

Theodore and Kost<sup>(4)</sup> found eight cases of primary meningococcal conjunctivitis amongst army personnel during an epidemic of cerebro-spinal meningitis. All cases were mild and responded to sulphonamides. Reid and Bronstein<sup>(5)</sup> describe a case of Group I primary meningococcal conjunctivitis in a two-year-old child. Allen and Erdman<sup>(6)</sup> record what appears to be the first case of exogenous meningococcal keratoconjunctivitis to be treated by penicillin—20,000 units were given by intramuscular injection three-hourly for five injections and local applications were not employed.

The present case showed no unusual features, but it is reported because no reference to previous cases was found in Australian literature nor in the records of the Children's Hospital, Melbourne.

### Clinical Record.

A female infant, aged seven months, had had a purulent discharge from the right eye for twenty hours. Delivered

by Cæsarean section at the eighth month of pregnancy, she had remained a healthy, bottle-fed baby without illness. She was an only child and the parents were healthy.

On examination the lids of the right eye were slightly swollen and congested, with purulent discharge exuding between them; the bulbar conjunctiva was slightly injected, the cornea was normal and the right preauricular gland was not palpable. Examination of the left eye and of her general physical condition showed no abnormality.

After smears and swabs had been taken the lids of the right eye were painted with 2% silver nitrate solution, which caused slight bleeding. Penicillin drops (1,000 units per millilitre) were ordered half-hourly and sulphacetamide drops (20% solution) four-hourly. The mother was instructed to protect the healthy eye and to isolate the baby from other children.

Next day the discharge had ceased, but there was excessive lachrymation and the bulbar conjunctiva had assumed a bluish-violet coloration. In view of the bacteriological report, sulphathiazole was administered orally—an initial 0.5 gramme being followed by 0.25 gramme four-hourly for twenty-four hours, when the drug was discontinued owing to vomiting. The right eye appeared normal within four days, but the penicillin and sulphacetamide drops were continued locally for six days. A conjunctival swab taken seven days from the onset of infection proved sterile. The child was afebrile throughout her illness and meningismus was absent.

Neither blood culture nor a complement fixation test was attempted. It seems unlikely that her illness was septicæmic in nature. The original conjunctival smear contained numerous polymorphonuclear cells with many extracellular and some intracellular Gram-negative diplococci. By agglutination they were found to be Group II meningococci.

In an attempt to determine the source of infection, naso-pharyngeal and conjunctival swabs were taken from the patient and from those who had been immediate contacts during the week preceding her illness. However, these were not taken until twelve days from the onset of symptoms.

Ox serum agar plates and sheep blood agar plates were inoculated and incubated in an atmosphere of 8% carbon dioxide. The contacts comprised father and mother, an uncle who had visited the family, and two friends who had taken care of the infant for an afternoon just prior to the illness. The uncle, who was not a frequent visitor, and who was known to have kissed the baby twenty-four hours before she became ill, was found to harbour Group II meningococci in his naso-pharynx. Group II meningococci were isolated from the patient's naso-pharynx and Group I meningococci from one of the friends.

Subsequent treatment, which was carried out by Dr. Mostyn Powell, consisted of a five-day course of penicillin nose drops and sulphamerazine given by mouth. A further naso-pharyngeal swab, after an interval, failed to yield meningococci.

#### Comment.

It was of interest to find that, although the culture was no longer grown from the eye after a cure had been obtained, meningococci were still present in her naso-pharynx.

There was no history of coryza just prior to the onset of the conjunctivitis, an occurrence which has been noted in some cases. It seems possible, although swabbings were not taken from contacts immediately, that our patient was infected by her uncle. A seven-months-old baby has but little contact with the outside world, and her opportunities for meeting meningococcal carriers would be limited.

Meningococcal disease was not prevalent in the community at this time, and both parents were free from infection at the time the examination was made.

This case illustrates the importance of cultural methods in the examination of conjunctival discharges. Too frequently a diagnosis of gonococcal conjunctivitis is based upon the findings in a smear stained by Gram's method. Perhaps primary meningococcal conjunctivitis is considered

rare only because cultural methods are almost universally neglected and because the condition responds readily to curative treatment.

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- <sup>(7)</sup> A. Cantor, H. A. Shelanski and C. Y. Willard: "A Microscopic Alkali-Solubility Test for the Identification of Gonococcus Colonies", *Journal of Bacteriology*, Volume XLIV, 1942, page 237.

## Reviews.

### HORMONES AND BEHAVIOUR.

IN "Hormones and Behaviour" Professor Frank A. Beach, of the Department of Psychology, Yale University, has collected results of observations and experiments carried out during recent years on various members of the animal kingdom.<sup>1</sup> The range of Professor Beach's survey extends over reversal of bisexuality in mating behaviour, aggression and submission in sex behaviour, conditioning, moulting, locomotor activity, migration, developmental changes in the endocrines, and variations in activity due to cycles of somatic responsiveness, to mention only a few titles. The chapter on emotion, or general emotionality, is disappointing, and observations on man are covered in three pages. The work contains a vast amount of information culled from over eight hundred sources, but the clinician will find little that he can apply to his practice.

From a medical standpoint the book, of some three hundred and fifty pages, has many shortcomings, but the experimenting zoologist and psychologist will find it of value for reference purposes.

The author concludes that major problems concerning the relationship between hormones and behaviour are amenable to experiment and that better understanding of similar effects in the human will result.

### THE AMERICAN MEDICAL ASSOCIATION.

WHEN the time came to think of celebrating its centenary the American Medical Association made a wise choice in selecting Dr. Morris Fishbein to compile a suitable volume of records by way of commemorating the great occasion. As the result of his sustained efforts a weighty, handsome and thoroughly satisfying publication, "A History of the American Medical Association, 1847 to 1947", extending to over twelve hundred pages, has been handed over to us and to all posterity in large, clear print.<sup>2</sup> Dr. Fishbein is well known as the efficient editor of *The Journal of the American Medical Association* and as the entertaining writer of many popular books dealing with the whimsicalities of orthodox medical practitioners and of "lesser breeds without the Law". In this volume he has requisitioned the services of a number of different authors to assist in presenting the whole picture and each has made a useful contribution in the section allotted to him.

<sup>1</sup> "Hormones and Behaviour: A Survey of Interrelationships between Endocrine Secretions and Patterns of Overt Response", by Frank A. Beach, with a foreword by Earl T. Engle; 1948. New York and London: Paul B. Hoeber Incorporated. 9½" x 6½". pp. 384. Price: \$6.50.

<sup>2</sup> "A History of the American Medical Association, 1847 to 1947", by Morris Fishbein, M.D., with the Biographies of the Presidents of the Association by Walter L. Biering, M.D., and with Histories of the Publications, Councils, Bureaus and other Official Bodies; 1947. Philadelphia and London: W. B. Saunders Company, Limited; Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9½" x 6½", pp. 1234, with many illustrations. Price: 70s.

The early beginnings of medical organization both in Great Britain and in the United States of America were prompted by a realization that little of real value could be accomplished for an improvement in the status of medical practitioners, in medical education, in relations with the community and the State authorities unless the profession could come together in a spirit of goodwill, inspired with the high purpose of extending knowledge of the medical and allied sciences, of maintaining the honour and interests of the craft and working for the introduction of preventive measures in helping to promote public health.

The practitioners of Great Britain in the early thirties of last century and of the United States of America in the late forties were fortunate in being able to secure the leadership of a man of character, vision, ability and untiring energy, whose chief mission in life was to bring the brethren up out of the wilderness and to give them a glimpse of the promised land. The work of Charles Hastings and Nathan Smith Davis in tilling the virgin soil and sowing the seed was carried out under conditions of striking similarity; and the mighty influence wielded by the two medical associations in modern times is due in no small measure to the splendid quality of those initial efforts. In a few particulars the methods employed in building up the organization and dealing with immediate problems were essentially different, but the basic objectives envisaged by the founders were substantially the same: to put an end to the eternal wrangling among members of the profession, to bring them together as a corporate body having a national representation and welded by a constitution strictly framed upon democratic lines.

The centenary book is to a large extent a cooperative effort, and it opens with a biographical sketch of the illustrious founder, Dr. Nathan Smith Davis. Then Dr. Fishbein gives a thorough survey of the American Medical Association from year to year throughout the century of its existence, quite naturally incorporating the history of the journal for the reason that its "activities were so completely integrated with the progress and life of the Association". At the conclusion of this, the most important section of the book, the editor is responsible for a section giving short biographical notes of the chosen few who have been honoured with the Distinguished Service Medal of the Association for outstanding work in medical science, and the list of recipients includes such familiar names as Rudolph Matas, Chevalier Jackson, Elliot Joslin and George Minot. The next section covering 260 pages is occupied with separate portraits and biographies of all the past presidents, some having a special interest for us: John Collins Warren (of anaesthetic fame), Samuel D. Gross, Alfred Stillé, James Marion Sims, Henry I. Bowditch, Austin Flint, the Mayo brothers, William Gorgas, J. B. Murphy and the founder, Dr. Davis, who occupied the presidential chair in 1864 and 1865, and lived to serve the association in many useful capacities for over fifty years. And it is interesting to find in the earlier part of the book that Oliver Wendell Holmes took an active part in the affairs of the newly formed association, acting as chairman of the committee on medical literature.

Finally, there is an important section in which different authors give a full account of the various councils and bureaux set up in connexion with the American Medical Association, which came into existence as its work continued to extend and become more specialized. It is obvious that many of these special departments, such as the Council on Pharmacy and Chemistry, function most efficiently for the benefit of the association and for the protection of the public. The last pages are taken up with short accounts of the various scientific publications sponsored by the association, which include the extremely valuable "Quarterly Cumulative Index Medicus" and the popular magazine *Hygiea*.

The book is well provided with portraits of medical practitioners who have figured prominently in the life of the association, and the frontispiece portrait of the founder is particularly striking. There are numerous typographical errors which are irritating when they occur in the giving of dates; but the *British Medical Journal* was not founded in 1865, and it is disturbing to see the name of John Abernethy consistently spelt "Abernathy" because, after all, he was an exceedingly good surgeon. Then it is stated on page 571 that Dr. Austin Flint (1884) originated the binaural stethoscope, whereas this useful instrument was introduced by Williams and modified by Leared and Camman in the early fifties.

This ambitious publication constitutes a fine record of achievement in the medical world, and the people of the United States of America have every right to feel proud of the men and women who have striven so hard throughout

the century to maintain high standards in the practice of their profession. The position was admirably summed up by the incomparable Dr. William H. Welch when he became president in 1910, and his words of wisdom have a deep significance for us today:

Organized effort is a distinguishing mark of modern civilization. It is as essential for the advancement of science, of education, of social and industrial reform, of philanthropic endeavour as for the promotion of commerce. With the remarkable progress of medical science, especially during the last three decades, man's power to control disease has been vastly increased and the sphere of usefulness of the physician has been correspondingly widened and with advancing knowledge will continue to expand. . . . Among the organized forces for advancing the prosperity, the happiness and the well-being of the people of this country, the American Medical Association has an important part to play. We are justified in the confidence that, with the united support and loyalty of the profession, this Association, broadly representative and standing for the best ideals of medical science and art and for professional and civic righteousness, will contribute a beneficent share to the working out of our national destiny.

It is to be hoped that Dr. Morris Fishbein will consider publishing a greatly abridged edition of this monumental work so that all may read and learn of the wonderful progress and development of this colossal organization.

#### RECENT ADVANCES IN SEX AND REPRODUCTIVE PHYSIOLOGY.

In the seven-year interval between the second edition of "Recent Advances in Sex and Reproductive Physiology" reviewed in this journal on November 30, 1940, and the present third edition an enormous amount of investigation has been carried out, but, as Buckle remarked about metaphysics, there is "much movement but little progress".<sup>1</sup> The author candidly admits in his latest preface that "no very striking advances have been made in the field of the Sex Hormones". The expert who can read through and attempt to assimilate such a mass of evidence, often of a conflicting character, is to be admired and perhaps pitied. In this latest edition special attention has been given to the metabolism of the steroids and especially the oestrogens and to problems of practical importance such as sterility. Dr. Robson is a pharmacologist, and to that fact we may ascribe the admirable handling of the various preparations available and their clinical use. This portion of the book will appeal not only to the gynaecologist but to the general practitioner to whom are too frequently addressed works of voluminous detail and formidable scholarship. It is not the fault of the author, but of the subject, that so many qualifications are discoverable in the text. There is hardly a page without some expressions like "probably", "on the other hand", "on the whole", "the evidence rather suggests", "future research will disclose", "seems likely", "not supported by later research" and many others of similar type. It has been the unkind fate of reproductive physiology to have hypotheses put forward which seemed convincing and then for these to have been blown sky-high by later findings. For example, the discovery of oestrogen and its action on the female was welcomed as a delightful explanation of the mechanism of female puberty and oestrus; but soon came the disconcerting announcement that oestrogen is recoverable from the urine of the man and in huge quantities from the urine of the stallion. Or take another example—implantation of anterior pituitary tissue into immature animals brings about sexual maturity. In the female all the signs of oestrus are observed and mating takes place. It was natural to infer that at puberty the pituitary begins to produce gonadotrophic hormones; but alas for this rather taking hypothesis, it was found that pituitary tissue from immature animals has precisely the same effect; actually anterior lobe taken from a fifteen-day-old rabbit precipitated oestrus in another young rabbit; whilst still more startling was the discovery that the pituitary of the fetal pig can do the same. What conclusion can we possibly reach here? Those readers of the chapter on menstruation who do not find their

<sup>1</sup> "Recent Advances in Sex and Reproductive Physiology", by J. M. Robson, M.D., D.Sc. (Leeds), F.R.S.E., with an Introduction by Professor F. A. E. Crew, M.D., D.Sc., F.R.S.; Third Edition; 1947. London: J. and A. Churchill, Limited. 8" x 5½", pp. 348, with many illustrations. Price: 21s.

minds enveloped in what John Bunyan called "the discouraging clouds of confusion" must be very few if indeed they exist at all. Still the author is to be commended for his vast reading and for his detached and critical analysis. Students no doubt prefer a dogmatic utterance to a lengthy presentation of pros and cons, but the researcher and the mature seeker after truth are glad to be given the facts with what guidance in forming an opinion a scholarly editor can offer. It is to these latter that the book can be recommended as conforming with the best traditions of the "Recent Advances" series.

### THE CHILD'S LUNG.

ACCORDING to Stefan Engel, the author of a new monograph, "The Child's Lung",<sup>1</sup> anatomy has always proved to be the basic science of medicine. Nearly half of his book is devoted to the developmental anatomy of the lungs, bronchi and bronchial glands, with measurements of casts of the bronchial tree and studies of post-mortem material at all ages from prematurity to puberty. This material is well presented with photographs, sketches and tables, but does not make very exciting reading except for anatomists.

The rest of the book is of more general interest to physicians and pathologists and contains good chapters on bronchitis and bronchiolitis, bronchiectasis, tuberculosis and pneumonia. There is an excellent section on tuberculosis of the bronchial glands, which appears to be the author's special hobby. The approach to these conditions is pathological; post-mortem material is studied, and on the basis of this and the known anatomical facts the aetiology is discussed. Thus the author shows from his own studies that whether multifocal pneumonia (bronchopneumonia) or unifocal (lobar) pneumonia complicates measles depends chiefly upon the age. Again, the conception that lobar pneumonia means the involvement of a whole lobe or greater part of a lobe may be true in adults, but it is a rare exception in children. Only the commoner diseases are dealt with, and for Australia at least there seems to be an undue emphasis upon childhood tuberculosis.

It must be stressed that this book deals, as stated in the title, with the child's lung, and not with the children suffering from pulmonary diseases, and that symptomatology and treatment find no part in it. It contains much useful material and some provocative viewpoints, but it is essentially a reference work for the medical library and not for the ordinary practitioner's bookshelf.

### OBSTETRICS.

THE problem confronting the author of any synopsis is to present within a small compass the essentials of his subject, and in so doing to preserve a balance between considerations of maximum and minimum importance. In the third edition of his "Synopsis of Obstetrics" Litzberg presents an interesting volume which is generally concise, but not to the point of brevity.<sup>2</sup> The author acknowledges that it is no substitute for a more elaborate textbook, but is presented as a syllabus of obstetric knowledge and practice.

In the management of normal pregnancy it is of interest to note that X-ray examination of the pelvis is advocated at the first examination of every pregnant woman, whereas gain in weight is not qualified by any reference to a reasonable limit of normality—surely a reversal of relative importance of clinical aids. The section on the Rh factor has been completely rewritten and is found somewhat unsatisfactory and confusing. One questions the statement that "either negative or positive blood may be used" for transfusion of an erythroblastic baby.

Concerning labour and its complications, whatever opinion may be held concerning the place of rectal examination, the author sets out clearly the information to be obtained on pelvic examination. No mention is made of conserving blood to the fetus by waiting for the cord to stop pulsating. The statement that Cesarean section is rarely required in

cases of brow presentation is not generally acceptable, but one agrees that watchful expectancy is the key to successful breech delivery, with interference only on clear indications. Episiotomy is advocated, but local anaesthesia to the perineum is not mentioned. Medical complications of pregnancy are summarized adequately, and it is interesting to note that the treatment of acute appendicitis is confined to that of the disease only. The section on diabetes has been revised. Substitutional hormone therapy is mentioned, but no details are included.

The discussion on the toxæmias of pregnancy follows routine lines, with emphasis on the conservative treatment of eclampsia. The latter is hard to correlate with the use of manual dilatation of the cervix and version as a means of conducting labour in this condition. *Placenta prævia* is regarded as an indication for emptying the uterus by the most conservative method as soon as diagnosed. The author rightly states that "blood transfusion is the sheet anchor of safety" in this condition, but his criticism of expectant treatment and Cesarean section does not seem justified in the light of recent papers on this subject.

A book of this kind has great possibilities. Some sections of this synopsis are very good, some perhaps outmoded and a little involved. It is, however, easy to read and should be of value to the practitioner rather than the medical student.

### BIOLOGICAL STANDARDIZATION OF VITAMINS.

IN the second edition of "The Biological Standardisation of the Vitamins" Dr. Katherine Coward has mainly considered the biological assay involving mammals and birds as test animals of the vitamins A, B, C and D.<sup>1</sup>

Determinations by physical characteristics and by colorimetric methods are dealt with in the case of vitamin A and some chemical estimations of vitamin C are mentioned. The vagaries and pitfalls in biological assays are brought out very clearly and will help to guide the less experienced worker through this field.

In addition to the experimental sections the author introduces the student to the elements of statistical analysis.

The value of the book lies in the fact that the technical details given come from one who has wide experience in biological assay of vitamins combined with sound criticism which is so essential even in a field where mathematics has the last word.

### CLINICAL METHODS IN SURGERY.

As stated in its preface, K. Das's book<sup>2</sup> on clinical methods in surgery is intended to guide the practitioner by answering the question: "How shall I investigate this case?"

It was produced in Calcutta during the year August, 1946, to August, 1947, a year which was a fateful one for that city. Continuous disturbances and restriction of outdoor movement put a limitation to the choice of material which could be incorporated. It is therefore very remarkable that the book has attained such a high standard. It is written in "note form", and the author endeavours to lead the student of surgery through his subject, having always in mind the practical viewpoint of a surgeon confronted by his patient. The illustrations in this book are perhaps its weakest part, owing, no doubt, to the production difficulties already mentioned. Many interesting conditions were photographed, but a standard of clarity was not at all times maintained.

In the section on "the acute abdomen" no mention is made of the acute gynaecological conditions which may give rise to an acute abdominal emergency—a ruptured ectopic pregnancy, ovarian cyst *et cetera*. Many other sections of the book appear to be likewise incomplete.

This book, published by one of our colleagues in a new "Sister Dominion", with whom we will no doubt have an even closer medical liaison in the future, deserves attention by all who seek to further their knowledge of procedure in surgical diagnosis.

<sup>1</sup>"The Child's Lung: Developmental Anatomy, Physiology and Pathology", by Stefan Engel, M.D.; 1947. London: Edward Arnold and Company. 8½" x 5½", pp. 340, with many illustrations. Price: 40s.

<sup>2</sup>"Synopsis of Obstetrics", by Jennings C. Litzberg, B.Sc., M.D., F.A.C.S.; Third Edition; 1947. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 7½" x 4½", pp. 416, with many illustrations, some of them coloured. Price: 41s.

<sup>1</sup>"The Biological Standardisation of the Vitamins", by Katherine H. Coward, D.Sc.; Second Edition; 1947. London: Baillière, Tindall and Cox. 8½" x 5½", pp. 232, with illustrations. Price: 16s.

<sup>2</sup>"Clinical Methods in Surgery", by K. Das, M.B. (Cal.), F.R.C.S. (England and Edinburgh); 1947. Calcutta: Hilton and Company. Sydney: Angus and Robertson, Limited. 9¾" x 7", pp. 250, with many illustrations.

## The Medical Journal of Australia

SATURDAY, MAY 15, 1948.

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### CARDIOLOGY IN AUSTRALIA.

THE visit of Dr. William Evans to the Royal Melbourne Hospital Centenary scientific session was an unqualified success and showed British medicine at its best. Dr. Evans is the third in the line of world-famous cardiologists to be in charge of the cardiac department of the London Hospital. His predecessors were Sir James Mackenzie and Sir John Parkinson; his lectures and his ward demonstration showed that he is true to their traditions.

It would be difficult to put a true value on the influence of these lectures on the members of an audience who came even from beyond the confines of Victoria. Everyone must have been aware that they were gathering the fruits of an inquiring mind turned to the study of simple everyday problems, of heart sounds, heart murmurs and heart pain, which were the subjects of three lectures. Dr. Evans reawakened by precept in his lectures and by example in his clinical demonstrations, interest in the auscultation of the heart. His principle of self-catechism exemplified in asking oneself whether, on auscultation, one hears two sounds or more, and whether in addition there are murmurs, draws attention to the need firstly for concentration when listening, and secondly for intellectual integrity. These disciplines have grown lax over the years, as the stethoscope has gradually fallen into disrepute. It is always important to be absolutely clear about the sounds that are heard and to attempt to weigh their significance. Physical signs, however, are of direct value only if they aid in diagnosis and in prognosis and thus in the proper management of a real or supposed illness; they may be of indirect value if they lead to research which throws light on the structure or function of the organ under observation. The path of clinical medicine is paved with physical signs which, though they may exist, are yet of no importance. Thus the mere description of physical signs, even though complete with details of the circumstances which surround them, is no guarantee that they are worthy of commemoration.

In discussing heart sounds, Evans<sup>1</sup> traverses a subject which has attracted the interest of French physicians for over a hundred years, from Laennec (1826) and Charcelay<sup>2</sup> (1838) culminating in the astonishingly accurate observations of Potain (1894). Evans for very good reasons objects to the term "gallop rhythm", so rightly connected with the name of Potain. Indeed, in his desire to record all forms of triple rhythm it seems that he fails to stress the outstanding importance of Potain's gallop rhythm. This presystolic triple rhythm indicative of left heart failure carries with it a significance not to be gathered from any other type in Evans's classification. It is an example of the true worth of a physical sign. It maintains its importance through the years on its own merit. When recognized, it is possible to state on this evidence alone that the patient has left heart failure just as surely as the discovery of the characteristic diastolic murmur denotes the presence of aortic incompetence. The same value cannot be attached to the triple rhythm of right heart failure, or of thyrotoxicosis, or of atrial septal defect. But after all, Evans did not set out to describe the gallop rhythm of Potain, but rather to set down his observations on all forms of triple rhythm, and to record and explain with the aid of electrocardiograms and phonocardiograms the presence of three (and sometimes four) heart sounds heard under varying conditions.

In a similar fashion Evans<sup>3</sup> has faced the problems of heart murmurs. He has recorded the murmurs by auscultation, investigated them by phonocardiography and then assessed their station in life. He discards the term "functional" (murmur) and substitutes the term "innocent" (murmur) for a murmur in the mitral region which in his opinion is not due to organic disease. He does not seek to explain the mechanism of the murmurs he describes, but rather to set down those features whereby they may be recognized as innocent. The term "innocent" may be misleading if it is used as the opposite to "organic" in the description of murmurs. It goes too far to suggest that, because a person has a systolic murmur with Evans's innocent characteristics, it cannot be due to structural abnormality or organic heart disease. The fact that a person with an innocent murmur and a history of attacks of rheumatic fever lives for years a life of unhampered activity is not to say that the murmur is not associated with the presence of rheumatic heart disease. It is within the experience of all physicians that a group of patients with rheumatic heart disease and mitral stenosis survive all the vicissitudes of a full life without serious cardiac symptoms. In regard to such people one might speak of an innocent presystolic murmur. The fact is that, unlike the presystolic triple rhythm of Potain, the systolic murmur cannot stand alone on its intrinsic merits. One judges its innocence or malignancy by the presence or absence of cardiac symptoms, and of cardiac signs such as heart size, other murmurs and the presence or absence of congestive heart failure, and adds to this often enough radiological examination and electrocardiography. Even in the absence of these diagnostic refinements, the occasions must be rare, in this country at least, when a person is laid aside and denied a full life because a

<sup>1</sup> British Heart Journal, Volume V, 1943, page 205.

<sup>2</sup> Archives générales de médecine, Third Series, Volume III, 1838, page 393.

<sup>3</sup> British Heart Journal, Volume IX, 1947, page 1.

systolic murmur is detected in the heart. Indeed, before Evans's visit the pendulum had swung too far the other way and systolic murmurs and accurate auscultation of the heart tended to be disregarded.

This leads to a discussion on some aspects of the possible development of cardiac departments in the hospitals in Australia. The history of these special units is that they have very often been built around an individual, and this rather than other factors determines the order in which specialties spring up in different hospitals. Though this process of evolution may not meet with the approval of those who plan the economy of hospitals and medical schools, it is likely to continue. If this is so, then it is essential that the specialists and their patients should be brought into the scheme of undergraduate teaching. They will of necessity be an essential part of post-graduate teaching. It must be apparent from Evans's visit that a cardiac department, if it is set up, must be something more than a place where an X-ray apparatus and an electrocardiograph are available for the examination of those suspected of heart disease. Even if Evans was not known by his writings, it was apparent to all that he had built his knowledge and gathered his wisdom from an enormous amount of work in his own field—work which is detailed and must often be tedious and time-consuming. Not satisfied to accept the authority of others, he has done research work on vasodilators, on the efficacy of various preparations of digitalis, on the course of the oesophagus in health and disease, and on electrocardiography and on phonocardiography. It is obvious that such a special department cannot be built up by a physician who visits twice a week. There must in addition be others working full and part time and for adequate remuneration. The reason that justifies the formation of a cardiac department is that it offers an opportunity to people with special aptitude and training to pursue ideas and observations which, though they may come to the general physician, he has not the time, the facilities or the number of specialized cases to follow up. There are certain disadvantages attendant on cardiology which are not so obvious in other departments of medicine. The first is that it can easily become a very confined field of work dealing with irreversible stages of disease. Too often the physician is presented with a *fait accompli*, and he bends his endeavours to the study of electrocardiography in the cold gradations of decay in the shape of coronary arteriosclerosis. Contrast, too, his comparative helplessness even after he has laid down the criteria for the diagnosis of early mitral stenosis with the prospect of restored health, when early pulmonary tuberculosis is diagnosed. Again, he may alleviate heart failure, but the writing is on the wall. Even with radiology and electrocardiography he cannot be precise in prognosis. It is still true that the heart knoweth its own bitterness. It is unfortunately possible to practise cardiology in a general hospital and to be virtually unaware of the problem of acute and subacute rheumatic infection and its cardiac manifestations if children under fourteen are debarred. It is timely to remember that the cardiologist (as do other specialists) draws his material with a very wide net. The greater his skill and repute, the more specialized his work becomes. In this way it is possible for a false impression to be gathered as to the true stan-

dard of work done in his own field by the general physician and the general practitioner. In this country the standard is high. In America a review of people rejected for war service on account of heart disease was made by groups of cardiologists. They confirmed the high degree of accuracy achieved by the ordinary medical boards without the advantages of special investigations. Yet, when all is said, it remains true that the development of cardiology has done much for human suffering and has still much to do. The recent developments in the understanding and the treatment of congenital heart disease are a salutary reminder that fresh and undreamed conquests may fall to the cardiologist with energy and imagination.

## Current Comment.

### CARCINOMA OF THE STOMACH.

THE subject of gastric carcinoma is always recurring; it has been referred to frequently in these columns and will undoubtedly require many more references. While it is not the most commonly occurring form of cancer, it appears to head the list of cancers causing death in most parts of the world. The most recently available Australian statistics show that for some years the number of deaths attributed to cancer each year has been from 8000 to 8500, distributed approximately equally between the two sexes. The number of deaths annually attributed to cancer of the stomach and duodenum has been between 1750 and 1830, of which over three-fifths are among males. Thus cancer of the stomach and duodenum is considered to be the cause of 20% to 22% of the total deaths attributed to cancer among the general Australian population, and of about 26% of those among the male population. According to O. H. Wangenstein<sup>1</sup> the death rate for cancer of the stomach is high in almost all countries, and deaths from gastric cancer comprise from 25% to 40% of all deaths from cancer. Of the 150,000 annual deaths from cancer in the United States, approximately 40,000 are due to gastric cancer. Herman Taylor<sup>2</sup> quotes from the Registrar-General's reports in Britain the fact that each year more than 13,000 people die from carcinoma of the stomach, but he does not state the total cancer mortality.

The point about these high relative mortality rates from cancer of the stomach is that they are quite out of proportion to the relative incidence of the condition. The general prognosis is poor. Wangenstein quotes the results of a number of different surgeons to justify his statement that it appears that 92% to 98% of patients who have gastric cancer will die of the disease within five years. Yet it has been calculated that, of patients who survive resection for gastric cancer for a period of five to seven years, the subsequent life expectancy is about the same as for normal persons. In other words, gastric cancer is curable, if recognized early enough, but a depressingly low proportion of subjects reach the surgeon in time. The twenty-first report of the British Empire Cancer Campaign (quoted by Taylor) indicates that in London five out of every six patients referred to hospital are beyond cure; the one patient in six whose growth is removed "submits his life to a 30% risk, and if he survives the crisis he has half the normal expectation of life at his age. For every patient who achieves that goal eleven of his pilgrimage fall by the wayside." At Lankenau Hospital in Philadelphia in 1944, according to G. C. Engel,<sup>3</sup> the operability in reference to a curative operation was only 17%. The highest figures do not seem to offer more than a 25% hope of cure.

<sup>1</sup> The Journal of the American Medical Association, August 2, 1947.

<sup>2</sup> The Lancet, April 17, 1948.

<sup>3</sup> The Journal of the American Medical Association, November 15, 1947.

It is not to the point here to discuss further the effectiveness of treatment *per se*. Appropriate surgical technique has reached a high standard and it should not need to be said that every subject of gastric cancer submitted to operation should have the benefit of the most competent and thorough surgery available; and research into further advances in treatment is scarcely within the province of the average practitioner. The most important aspect of this whole tragic problem is related to ordinary everyday practice. As Engel points out, there are two reasons for the low rate of operability: difficulty in diagnosis and "the delay that takes place all along the line". The delay factor is essentially a human problem whether considered from the patient's or the doctor's angle. Essential ignorance is, of course, important. The ordinary person must be taught to recognize the warning lights and a good deal has been done and is being done along these lines. Taylor, a trifle diffidently, suggests the text of a letter which might be sent to every citizen on his or her fortieth birthday by the Minister for Health. This would probably be a controversial measure, but many will agree with his contention that such an approach would not create hypochondriacs, whom we have always with us. Whatever the measures a constant campaign for educating the lay public is important, and the medical profession, individually and collectively, has an obligation to give its support and active cooperation. An even greater obligation rests on the practitioner to maintain his own knowledge, for ignorance on his part cannot be excused in the same way; it is a heavy practical problem but part of that burden which every practitioner of medicine has voluntarily assumed though he seldom cares to think of it in terms of heroics. The human factor in the patient, who knows or suspects but procrastinates, was well discussed by N. V. Youngman in an article in this journal on May 10, 1947, and there is perhaps little to add to that. On the human factor in the doctor it may be as well not to moralize, but the hackneyed phrase is still true: "More is missed by not looking than by not knowing."

The further factor in delay is the provision of facilities for diagnosis and linked with this is the consideration of difficulties in diagnosis. Much of this is familiar ground and we will draw attention to only a few matters of particular interest. After discussing the personal and family history, Engel lists as the most important aids to diagnosis X-ray examination, gastroscopic examination, the fractional test meal, the determination of serum protein values and the complete blood count. To these might now be added the cytological method introduced by Papanicolaou. An encouraging report on its use as an aid in the diagnosis of gastric carcinoma has recently been made by Ruth M. Graham, Howard Ulfelder and Thomas H. Green.<sup>1</sup> These workers examined gastric fluid from 50 patients with gastric symptoms. Of 24 patients proved at exploratory operation to have gastric carcinoma, the results of examination of smears were positive in fifteen instances. Of seven patients with resectable lesions of the stomach, the smear results were positive in five cases. Of the remaining 26 patients, who did not have cancer, a positive result was reported from the smear in one case, this patient having a benign gastric ulcer. Of particular interest is the fact that the earlier the lesion, the greater appeared to be the accuracy of the method. Graham and her colleagues suggest that it may be of great value and place it beside mass radiography as an approach to the general diagnostic problem. The latter method has been strongly advocated by Taylor in the article in *The Lancet* already referred to, his principal contention being that there can be no real advance until the general practitioner can obtain a radiological report in every case of suggested cancer. His plan would require provision of equipment in suitable centres and he suggests that more use might be made of junior radiologists and technicians to cover the volume of work. This brings up the question of inadequate radiological investigation, about which V. J. Kinsella writes in strong terms in this issue; but Taylor's opinion cannot be lightly put aside that, in a situation where five out of six patients are lost from the start,

an imperfect return is better than none. Engel also advocates the more liberal use of X-ray examinations with other appropriate investigations and Wangenstein suggests the establishment of cancer detection clinics.

The thought in the background of all these discussions is that of the insidious onset of gastric cancer. The education of the public in the appreciation of classical symptoms, the ability of the general practitioner or of the specialist to recognize the established lesion—these do not meet the real problem of gastric cancer. Any statement on the prognosis of this disease is almost inevitably a paradox, as is seen from the history of D. F. D. Wilkie, the Scottish surgeon, who at a conference on cancer in 1928 stated, when speaking on "Early Diagnosis and Treatment of Gastric Cancer", that the technique of the operation for gastric cancer had been raised to a standard which left little to be desired; for, Wangenstein says, Wilkie subsequently died of a gastric cancer diagnosed too late. The crux of this problem is the need to establish a diagnosis before the clinical picture is clear and this, whether it involves cancer detection clinics, mass radiography, routine examination of gastric fluid for cells or other methods, is likely to need vigorous—and probably expensive—action.

#### NERVE LESIONS COMPLICATING CLOSED BONE INJURIES.

SERIOUS nerve injuries complicating closed bone injuries are not common and most surgeons see them too infrequently to gain a great deal of experience in their management. H. J. Seddon, Nuffield Professor of Orthopaedic Surgery at Oxford, has had rather unique opportunities in this regard at the Oxford Peripheral Nerve Injuries Centre where over 2500 nerve injuries of every kind have been seen during a period of six years. In a recent paper<sup>1</sup> he has discussed this type of nerve damage with particular reference to the upper limb. Three chief types of nerve injury are mentioned. He dismisses briefly the transient paralysis called neuropraxia with its usually complete motor loss, very slight sensory loss and absence of the reaction of degeneration in the muscles; recovery is usually complete before the conclusion of treatment of the fracture. The other two types are associated with traction and with violence acting transversely to the length of the nerve. The distinction is important as traction usually produces a much more serious lesion; a great length of nerve may be damaged as compared with the localized lesion caused by transverse violence and the prognosis for a persistent complete paralysis due to traction is grave. The clinical distinction between traction and direct injuries may be difficult, but Seddon describes one cardinal feature which is pathognomonic of traction injury: the indication by the extent of the paralysis that the lesion of the nerve is proximal to the site of the fracture. He then goes on to describe in detail 79 cases of severe paralysis resulting from closed bone injuries of the upper limb and draws certain conclusions. He considers that a good prognosis may be given with confidence for any multiple nerve lesion in the region of the shoulder that is not due to traction, though conservative treatment will need to be continued painstakingly and for a long period. Isolated circumflex nerve injuries are thought to be usually due to traction and the prognosis is uncertain. Fractures of the shaft of the humerus sometimes cause division of a nerve, usually the radial, but the surgeon may justifiably wait till the axons growing at the rate of one millimetre a day ought to have reached the most proximal muscle. If there is then no evidence of recovery from electromyographic studies, exploration is indicated. Nerve suture will not necessarily be undertaken even then, but it should not be delayed beyond one year after the injury. Professor Seddon has given us the fruit of a remarkable clinical experience in this paper and it is to be hoped that more will be heard of his work.

<sup>1</sup> *Surgery, Gynecology and Obstetrics*, March, 1948.

<sup>2</sup> *The Journal of the American Medical Association*, November 15, 1947.

## Abstracts from Medical Literature.

### PATHOLOGY.

#### The Benign Giant Cell Tumour of Tendon Sheaths.

ACCORDING to Lee N. Foster (*The American Journal of Pathology*, July, 1947), in its earliest phases of growth the benign giant cell tumour of tendon sheaths has a highly vascular structure that becomes disrupted by sclerosis, but proliferation of the intervacular and endothelial cells of the tumour continues, limited only by the degenerative tendencies inherent in these cells and to some extent by stromal overgrowth. These are features of autonomous growth and definitive of a true neoplasm. As the development of the tumour progresses, certain of its cellular elements phagocytose lipid and haemosiderin, while some coalesce with one another to form multinucleated giant cells. These activities represent behaviour characteristics of the reticulo-endothelial system, and since they are centred about blood vessels it is concluded that the tumour is a sclerosing haemangioma.

#### Focal Anaemia, Leucocytosis and Fatty Infiltration of the Liver.

IN a series of cases of certain yellowish spots (so-called "septic liver spots") frequently seen on the surface of the liver, which stand out clearly against the surrounding tissue, two quite different types of lesions were observed by Franz Wenger (*Archives of Pathology*, October, 1947). In the first the parenchyma under the spot was severely anemic, this lack of red blood cells being responsible for the clear colour of the whole area. In many cases there was, in addition to the anemia, a dense accumulation of polymorphonuclear leucocytes, lymphocytes and large mononuclear cells inside and outside the sinusoids. The second type, in most instances, was wedge-shaped on the cut surface and sharply limited; it showed severe fatty infiltration of the liver cells. No satisfactory explanation can be given by the author as to the causation and genesis; an inflammatory origin can be ruled out probably for both groups of lesions. In regard to the first, all the findings, including the local leucocytosis, might be explained by vascular spasm and consecutive slowing down of the blood stream during agony. The second group seems to have an embolic origin. As both types of lesions are associated with a great variety of diseases, the name "septic spots" should be replaced by "foci of anemia" or "foci of fatty infiltration" respectively.

#### $\beta,\beta'$ -Dichlorodiethyl Methylamine Hydrochloride.

THE effects of one of the nitrogen mustards,  $\beta,\beta'$ -dichlorodiethyl methylamine hydrochloride, on the blood-forming organs have been investigated in rabbits and dogs by C. R. Cameron, F. C. Courtice and Rosa P. Jones (*The Journal of Pathology and Bacteriology*, July, 1947). There is progressive necrosis of the germinal centres of the lymph glands and spleen, with polymorphonuclear cell infiltration, commencing as early as three hours after

the injection and progressing until the third and fourth day. The bone marrow shows at first congestion of the sinusoids and then a rapid depletion of the myeloid tissue from one to three days after the injection. Recovery occurs in about a week. The white cells of the blood are affected. Neutrophilia is observed during the first twenty-four hours, followed by neutropenia. The numbers of lymphocytes, basophile cells and eosinophile cells begin to fall within a few hours after the injection. The fall in the numbers of all cells is at a maximum on the third or fourth day, and from the fifth to the seventh day the numbers of all cells return to normal if the animal survives. The red cells are little affected by a single dose, but repeated injections for four weeks cause progressive anaemia. Collection of the thoracic-duct lymph in dogs shows that lymphocyte production is greatly reduced as the blood lymphocyte count falls. The production of neutrophile cells is also greatly decreased, as there is no neutrophile cell response to trauma when the bone marrow is damaged. The formation of new red cells is likewise decreased, as the normal reticulocyte response to haemorrhage is not observed when the neutrophile cell count is kept low by repeated injections, but is evident as soon as the number of neutrophile cells rises on cessation of the injections.

#### Spondylitis Deformans and Myositis Ossificans.

ACCORDING to Edwin F. Hirsch and James W. Riley (*Archives of Pathology*, November, 1947) bone spurs develop along the vertebral column in *spondylitis deformans* through enchondral ossification of proliferated fibrocartilage, activated to growth by the trauma of stress. The lesions of traumatic *myositis ossificans* evolve through a similar process of enchondral ossification in proliferated fibrocartilage. The bone spurs of *spondylitis deformans* develop along the vertebral column, in a sense, to compensate and strengthen ligaments weakened by continuous stress at their points of insertion. The bone growths of traumatic *myositis ossificans* are spurious growths, occurring in tendons or ligaments injured at their points of insertion. The fibrocartilaginous tissues stimulated to proliferate by injury are normal constituents of ligaments and tendons where these attach to bone. Accordingly, enchondral ossification occurs as a normal sequence of traumatic proliferations of fibrocartilage in the two disorders, *spondylitis deformans* and traumatic *myositis ossificans*, which have seemed to be entirely unrelated. Trauma is the accepted cause of each.

#### Pathological Significance of the Ductus Arteriosus.

ACCORDING to Lester S. Blumenthal (*Archives of Pathology*, October, 1947) the *ductus arteriosus* is a true muscular organ which on proper stimulation is capable of contracting strongly enough to obliterate its lumen. The stimulation is apparently the increase of the oxygen saturation of the arterial blood brought about by the onset of respiration in the newborn infant. After the contraction has been maintained for several days, actual histological changes take place. Muscle tissue is replaced by tissue of

a lower order of specialization. In some cases the replacement is slow, and no necrosis occurs; however, in others rapid degeneration with necrosis and calcification takes place. The cause of this tissue replacement and degeneration appears to be relative anaemia of the wall of the ductus brought about by contraction of the muscle. This contraction narrows the lumen, compresses the *vasa vasorum* and thus decreases the blood supply of the vessel wall. These tissue changes occurring in the *ductus arteriosus* as the result of anaemia seem to be similar to those observed in arteriosclerosis. This work has led the authors to the opinion that anaemia plays a large part in arteriosclerotic processes and may possibly be the fundamental mechanism through which the various causal agents bring about arteriosclerotic changes.

#### Fibrosis Uteri.

ACCORDING to R. Shoemaker (*Archives of Pathology*, December, 1947) the patient with *fibrosis uteri* may be described as having a history of profuse menorrhagia without metrorrhagia and usually without dysmenorrhoea. Her age may be any age between thirty and fifty-five years. She may be nulliparous or primiparous, but most commonly is multiparous. On pelvic examination the uterus is found symmetrically enlarged and in normal position. The excised uterus is revealed as a slightly to moderately enlarged organ. The myometrium is pale, finely textured and thickened, with consistency increased throughout or at least in its inner half. On microscopic examination an increase of fibrous tissue is observed in the submucous layer of the myometrium equalling or exceeding 15% of the width of this layer and appearing as an increase of both the interfascicular and the intrafascicular connective tissue. The author states that this study does not reveal the exact causation or pathogenesis of the lesion, but that it does provide ample evidence that it is not an end stage of chronic metritis, that hypertrophy of muscle fibres plays a minor role (occurring in only 11% of the cases), and that it is not a form of subinvolution of the uterus. *Fibrosis uteri* may be considered a fairly well-delimited clinical and pathological entity. It is different from simple muscular hypertrophy and subinvolution and establishes a defect of the myometrium as a causative factor in menorrhagia.

#### Reticulo-Endothelial Cells, Toxic Antigens and Infection.

OBSERVATIONS by P. Bueno (*Archives of Pathology*, December, 1947) show that certain cells of the reticulo-endothelial system are damaged when toxic antigens are introduced into the organism or during infection. Particularly interesting, however, is the fact that these are the same cells which react during the anaphylactic phenomenon. This shows that certain undifferentiated mesenchymal cells have a special capacity of reacting to different kinds of antigenic stimuli. On the other hand, the results confirm a series of researches which indicated that toxins damage lymphatic tissue. Observations by the author showed, moreover, that lesions occur not only in the germinal elements of the lymphatic tissue, but also in cells which

are considered as a source of monocytes or histiocytes (for instance, extra-follicular germinal cells of the cortex of the lymph glands and the Kupffer cells). Therefore, it is evident that there is a similar way of reaction among the precursors of mononuclear cells. Finally, it is interesting to consider that these findings, which demonstrate a kind of electivity of antigens for certain undifferentiated or germinal reticulo-endothelial cells, seem to accord with the concept that these cells function as producers of antibodies.

## MORPHOLOGY.

### Muscles of the Pelvic Limb.

H. A. HAXTON (*The Anatomical Record*, July, 1947) compares the muscles of bipeds and quadrupeds. He states that the most powerful muscles in the hind limbs of quadrupeds are the hamstring group, particularly the *biceps femoris*, and the gastrocnemius. In man's lower limbs the strongest muscles used for locomotion are the *gluteus maximus*, the quadriceps, and the soleus. The hind limbs of quadrupeds act mainly as propulsive levers, and as such require strong extensor muscles at the hip joints and strong flexors at the knee and ankle joints. Furthermore, there is no special provision in the hind limbs for the checking of forward momentum, since this is a function of the forelimbs. In the locomotion of man the lower limb acts chiefly as a propulsive strut, for which strong ankle flexors are important, and as a retropulsive strut to control the body's forward momentum. For the latter purpose powerful hip and knee extensors and ankle flexors are required. The principal mechanical stresses in the hind limbs of quadrupeds are best met by powerful muscles acting on two or more joints, while in man strong one-joint muscles are more suitable.

### The Hippocampus and the Sense of Smell.

A. BRODAL (*Brain*, June, 1947) has reviewed the literature on the anatomy and physiology of the hippocampus in mammals, particularly as regards its alleged association with the sense of smell. The olfactory fibres from the olfactory bulb and the anterior olfactory nucleus have not been traced to the hippocampus proper. They appear to end only in the peri-amygdaloid and prepiriform area of the so-called piriform lobe. The afferent fibres to the hippocampus arise mainly from the endorhinal area, small additional contributions coming from the posterior part of the cingular gyrus and probably from the *induseum griseum*. All the efferent fibres from the hippocampus leave the latter as the fornix. Some of its fibres end in the hypothalamus, the preoptic region and the septal areas and the habenula, but most of them go to the medial and part of the lateral mammillary nucleus. From this a massive pathway leads through the anterior thalamic nucleus to the cingular gyrus. From a structural point of view the hippocampus must be considered a typical effector structure, transmitting but modest proportion of impulses ultimately olfactory in origin, integrated with others the nature of which is not yet known, to

the cingular gyrus and to a lesser extent to the hypothalamic, preoptic and septal areas and the habenula. All the latter connexions are probably concerned in influencing subcortical reflexes, olfactory and others, as well as hypothalamic activity. The projection to the cerebral cortex is not understood from a functional point of view. It may be concerned in cortical control of autonomic functions. Recent physiological experiments have yielded no support for the conception that the hippocampus has important relations to the sense of smell in mammals, nor does clinical evidence seem to favour this view.

### Development of Human Auditory Vesicle.

T. H. BAST *et alii* (*The Anatomical Record*, September, 1947) report that in the course of a comprehensive study of the development and adult structure of the human ear and temporal bone, certain details have been added to the more familiar concepts of morphogenesis of the otic labyrinth. In the present communication further details are given particularly concerning the endolymphatic duct and sac.

### Adrenal Cortex.

R. G. HARRISON AND A. J. CAIN (*Journal of Anatomy*, July, 1947) have found in the adrenal cortex of the rat considerable variation in the quantity and distribution of lipine, of gross lipid, and of substances colouring red with Nile blue. No corresponding variation in cholesterol is seen; this substance appears to be present always in the *zona glomerulosa* and *zona fasciculata*, and absent or nearly so from the *zona reticularis*. After staining for lipoids is carried out, three main regions are distinguishable in the cortex. (a) The outer region contains the *zona glomerulosa* plus a small part of the *zona fasciculata*. (The latter may or may not be sudanophobe in part.) This region is very variable indeed with respect to lipoids. (b) The middle region is the heavily loaded part of the *zona fasciculata*. It corresponds to Bennett's secretory zone, but it is possible that lipoids other than steroids are secreted in the outer part only, and merely stored prior to dispersal in the inner part. (c) The inner region is the rest of the *zona fasciculata* plus the *zona reticularis*, and is usually poor in lipid, except in some of the cells of the *zona reticularis*. This corresponds to the post-secretory plus senescent zones of Bennett. Some of the variations observed are not correlated with age and sex.

### Normal Adult Human Erythrocyte.

P. H. RALPH (*The Anatomical Record*, August, 1947) states that the study of normal living blood cells *in vitro*, untreated or supravitaly stained and with the use of histochemical procedures, has revealed new facts of some significance and has made possible a more accurate interpretation of previously recorded observations. The untreated, uninjured erythrocyte in blood freshly drawn from a normal adult individual may be considered to approach closely the ideal of unaltered morphology and undisturbed physiological function. Studies following experimental procedures are of interest only in so far as they explain the

structure, behaviour or function of the cell *in vivo*. The author reports that the presence of a plasma membrane has been demonstrated microscopically for the mammalian erythrocyte. This membrane has been demonstrated to be (a), elastic, (b) subject to the action of enzymes in the plasma which are activated when the blood is extravasated, and (c) partially fixed or jelled by the action of citrates and oxalates. The membrane contains substances extractable by alcohol and acetone (lipids) which cannot, in their natural state, be stained by any lipid stain, but which after fixation (saturation of unsaturated bonds) by iodine, osmic acid or bromine can be stained with Sudan black. When hemolysis occurs the haemoglobin leaves the cell without creating any visible rupture in the plasma membrane. The assumption of stickiness by the red blood cell is not a contributory cause to coagulation. Very careful handling of blood is necessary if one is to avoid the formation of misleading artifacts. The mature normal erythrocyte has no visible internal structure or organelles. The immature erythrocyte may contain mitochondria and may develop, with proper treatment, "A granules", vacuole, and reticulum, all of which are separate entities and are considered to be evidence of primitiveness. The "thickness" and refractivity of the erythrocyte surface in dark field examination are directly proportional to the concentration of haemoglobin within it.

### Elastic Tissue of the Skin.

J. C. DICK (*Journal of Anatomy*, July, 1947) describes the distribution of yellow elastic tissue in the skin. He finds that the elastic tissue consists of two parts: large fibres in the deeper part of the dermis, and a fine network of small fibres lying close under the epidermis. Variations in the quantity and characteristics of these two parts are described and illustrated in skin from different situations of the body, and in skin from subjects of different ages. A brief description is given of the changes in the elastic tissue in subjects with oedema. The reticular fibres of the skin at the junction of dermis and epidermis have been shown to be distinct from the elastic tissue.

### Persistent Ischiatic Artery.

J. C. FINERTY (*The Anatomical Record*, August, 1947) states that persistent ischiatic arteries have been recorded in the literature sixteen times and show considerable variation in details, especially in the course and extent of the femoral artery, which in all cases appears to be represented. In the present case a persistent ischiatic artery is described in the body of an eighty-five-year-old male Negro which differs from previous descriptions in the presence of a typical, but small, femoral artery anastomosing with the ischiatic artery within the popliteal fossa. Venous drainage of the extremity is by a large ischiatic vein emptying into the femoral vein by way of a plexus within the *adductor magnus* muscle, by veins accompanying the femoral and obturator arteries and by normal saphenous veins. The presence of a normal inferior gluteal artery in this case suggests that the inferior gluteal artery may be derived from the hypogastric artery as an independent branch, rather than as a remnant of the original axial artery.

## British Medical Association News.

### SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on November 20, 1947, at Broughton Hall Psychiatric Clinic, Leichhardt. In the absence of Dr. S. Evan Jones the meeting was arranged by Dr. Broughton Barry and the medical staff of the hospital. Part of this report appeared in the issue of May 1, 1948.

#### Presbyophrenia.

A married woman, aged thirty-four years, had been born in Guyra and had never been outside New South Wales. She was the youngest of five children. There was nothing relevant in the family history, and she had worked for her living until her marriage six years previously. Her condition had been deteriorating over the last two years. She had lost interest in her home and had been sluggish in her movements and forgetful. She rambled when she talked. She had made no complaints and did not think that she was ill. She lay down a great deal. She did not perform her household duties and slept during the day as well as at night. (The foregoing history was obtained from her husband.)

On her admission to Broughton Hall, the patient gave a rambling, incoherent and disconnected account of herself, and said that she felt well. She showed considerable intellectual deterioration and her memory was impaired; she was voluble but incoherent, she did not comprehend simple questions and she was unable to calculate. Her blood pressure was 152 millimetres of mercury (systolic) and 90 millimetres (diastolic). The picture was that of a rapidly progressing dementia starting very early in life. The diagnosis of presbyophrenia was made—an organic dementia of obscure aetiology. Neurosyphilis was excluded by the negative serological findings. There was one other remote possibility—Huntington's chorea. The patient's maiden name was that of a family which had produced thirty or more cases in three or four generations. However, it had not been possible to connect the patient with that family. Dementia might occur without chorea in Huntington's chorea.

#### Presbyophrenia with Possible Pick's Disease.

The eighth patient shown was a casual labourer, aged fifty-nine years, who had been born in England and had come to Australia at the age of twenty-four years. Both parents had died in England in their sixth decade, the causes being unknown. The patient for the last two years had shown progressive mental deterioration, the process increasing rapidly in the last few months. He had continual headache, his memory began to fail, he became emotional at times and he lost his way easily. Six months previously he had been admitted to the Royal Prince Alfred Hospital. The cerebro-spinal fluid gave negative results to all tests. An electroencephalogram was prepared and the Rorschach test was carried out; the report stated that, if anything, the results confirmed the diagnosis of Pick's cortical atrophy. On his admission to Broughton Hall the patient was confused, foolish, euphoric and disorientated, and his memory was grossly impaired; he could give no satisfactory account of himself. His blood pressure was 174 millimetres of mercury (systolic) and 98 millimetres (diastolic).

#### Possible Cerebral Neoplasm.

A male patient, aged forty years, had had a stroke in 1945, in which without warning he fell to the ground without losing consciousness; he was paralysed on the left side of his body, but after four days he began to regain power. He noticed that the toes of his left foot were "bunched"—bent downwards and drawn together—and this attitude still tended to recur. Soon after the stroke the patient found that he was over-sensitive to heat and cold on the left side of his body. The contact of a fly on the left side of his face was painful, and he experienced discomfort while shaving. He had become excessively emotional; he became violently angry—"disgustingly so" was the expression he himself used. The left upper and lower extremities ached continually and became more rapidly fatigued than the right. About a year later he had another seizure in which he became unconscious. Examination revealed paresis of the left side of his face, his speech was thick and his pupils were unequal but responded to light; hyperæsthesia and hyperalgesia of the left side of the body were present.

Plantar stimulation on the left side produced tonic plantar flexion and adduction of the toes, which tended to persist and suggested a plantar group reflex. The blood pressure was 140 millimetres of mercury (systolic) and 88 millimetres (diastolic). No abnormalities were detected on investigation of the blood serum and the cerebro-spinal fluid; the pressure of the latter was normal. No changes were detected in the fundi.

The patient became depressed and irritable and was discharged from hospital on December 24, 1946; he left fully confident that his depression would go and that he would be able to control his emotions. He found that this was not so; he had an argument with his wife and abused her to the extent of imperilling his domestic relationship, and returned to Broughton Hall. Shortly afterwards he lost the power in his left leg for a day and dragged it while walking; power soon returned. For a few days he suddenly lost his pain and emotional disturbances and felt much better, but the improvement was not maintained.

The comment was made that lesions in the ventral part of the optic thalamus might produce the syndrome of Dejerine and Roussey, characterized by crossed hemianæsthesia and hemianalgesia, subjective intractable pain on the affected side, excessive emotional responsiveness to stimuli, chorioathetosis and hemiataxia, and hemiparesis from involvement of the pulvinar; all these symptoms were not necessarily present. The condition of the patient under discussion conformed to this syndrome, but lacked hemianæsthesia, athetosis, ataxia and hemianopsia. The most common cause of the thalamic syndrome was a vascular lesion, hæmorrhage, thrombosis or embolism. It was sometimes caused by inflammatory conditions or tumours. In the condition of the patient there was nothing to suggest a vascular lesion, although it could not be excluded. Neurosyphilis was excluded by the negative serological findings. A provisional diagnosis of neoplasm in the region of the right optic thalamus had been made, although there were no signs of increased intracranial tension. The apoplecticiform attacks were possibly due to hæmorrhage into the neoplasm.

#### Congenital Neurosyphilis.

The next patient was a male, aged sixteen years, the only child of his mother's second marriage; he had two elder half-brothers, both of whom were healthy. The father had suffered from "gonorrhoeal rheumatism" when the patient was aged eighteen months, and the mother left him, taking the child. The mother first noticed symptoms when the child was aged ten years. He began to grow "sheepish", and hunched his shoulders as if afraid. The symptoms gradually spread; he became irritable and cranky, yawned excessively and became restless and troublesome. Then he had a seizure and lost the use of his legs and the sight in his left eye; the symptoms were temporary and function returned after a few days. Subsequently his behaviour degenerated further; he threatened suicide, and began to show sexual aberrations and to take too much notice of little girls. He was admitted to the Royal Prince Alfred Hospital and treated for cerebral syphilis with "Mapharsen", bismuth salicylate and an intensive course of penicillin. He was admitted to Broughton Hall from the Royal Prince Alfred Hospital. On his admission he was seen to be eunuchoid and asthenic; his legs were sabre-shaped and he had Hutchinson's teeth. Gross intellectual deficiency was present and he was dull and childish. The cerebro-spinal fluid yielded a positive response to the Wassermann test and was found to contain no cells; the Takata-Ara test produced no reaction, a slight amount of globulin was present, and the response to the gold sol test was "1122200006"; this indicated that the process was "burnt out".

#### Hysterical Somnolence.

The next patient was a female, aged sixteen years, the third of seven children. The mother was over-protective and dominating and the patient had widespread psoriasis. Her present condition had appeared about eleven months earlier, when she had a period of "sleepiness", during which she seemed heavy but could be roused. She had had recurrent attacks since, usually at the catamenia. The somnolence might last for two or three days, and on the last occasion she could not be roused for two days. During the attacks she did not pass urine, did not eat and remained lying in one position. When forcibly moved she cried hysterically. The patient said that the first warning she had of an attack was the occurrence of pain in the iliac fossæ, and then she became drowsy "like going under an anæsthetic"; she did not remember what happened during her sleepy attack. She was admitted to Broughton Hall on September 12, 1947. On September 23 she complained of

abdominal pain and became drowsy; she could be moved by painful stimuli, but made no response. This attack lasted for three days, during which she did not eat and did not pass urine.

The comment was made that the patient was suffering from so-called hysterical somnolence; she did not actually sleep, but was in a state of abstraction and withdrawal, and obviously resented any attempt to rouse her. It was of interest to note that the first attack followed a vaginal examination under "Pentothal" anaesthesia (the onset of an attack was "like going under an anaesthetic").

#### Manic-Depressive State.

A female patient had been separated from her husband eight months earlier; she had one child, aged eight years. She had had a normal childhood and had worked as a shop assistant for five years prior to her marriage at the age of twenty-one years. Marital relations were unsatisfactory; the patient was always frigid, and the husband was cruel and had been "bound over" for two years for assaulting his wife. There were financial worries. The patient had been ill for a year; she was worried about her marital and financial position. She had become very religious and wanted to be baptized; she was depressed and cried a lot, lost interest in everything and wanted to lie down all the time. In September, 1947, she became restless and excitable, saying that she could hear and see things that were not there and smell odours. She settled down after a few weeks and was admitted to hospital on October 3. When interviewed she admitted her delusions and hallucinations of six weeks previously, said that they were no longer present, and appeared to have good insight into her condition. She was depressed, but otherwise fairly normal. She was given electro-convulsive treatment, but remained in this quiet, depressed state until November 6, when she became emotional and restless. On November 9 she was in a hypomanic state, aggressive and abusive, and saying that she was being poisoned and that the medical officer was breaking her legs every day because she had committed adultery (there was no record of this).

#### Paraphrenia.

A female patient, aged thirty-six years, had three children. She was the youngest of a family of eight, and was supposed to have had "nervous trouble" in childhood; this might have been chorea. She did not agree with her father and lived away from home. She had married at the age of twenty-four years; *coitus interruptus* was practised, and she never received satisfaction from intercourse. For two years she had been abnormal and had expressed the belief that her husband was unfaithful; this was unfounded. She then said that people were flashing messages to her husband. She began to bark at the dogs belonging to the people next door. She lacked judgement in managing the household affairs, wasting money and lying down for days doing nothing.

On her admission to hospital the patient said that she was a "substitute wife" for her husband, that she liked children, but there were plenty of reasons why she did not have more. She refused intercourse whenever possible, and said that people were interfering with her from a great distance and that she was going to have a magic baby: "I've been impregnated by somebody I don't know half a mile away." The patient's consciousness appeared clouded and her attention poor. She was garrulous and irrelevant in her conversation and was engrossed in her own delusional world.

A second patient suffering from paraphrenia was a female, aged thirty-one years, who had been admitted to Broughton Hall on October 14, 1947. She had one brother in hospital in South Australia (possibly Parkside Mental Hospital). Her infancy and childhood had been normal. She had been married for four years and had two children. Her husband said that she had been mentally abnormal for the past five months. She had begun to imagine that people were walking round the place at night talking about her. She had improved for a while after moving to another State, but again relapsed, thinking that people were talking about her; they said that she was no good, that her breasts were too small. She thought that her children were to be taken away from her. She became depressed and tried to shoot herself. Since her admission to hospital she had been acutely hallucinated; she had ideas that people followed her about with an electric machine and talked to her from the machine, telling her to shoot herself. She had been found with a dressing-gown cord round her neck. She had put her foot through the window of her room. She had expressed the fear that she would be murdered by men in the laundry, and that men were threatening the medical officer's life. The

comment was made that this patient had an active paranoid psychosis; she was noisy and restless and had made two suicidal attempts in an endeavour to avoid her persecutors.

A third patient suffering from paraphrenia was a female, aged forty-one years, the wife of a postal clerk. Her mother was a nervous woman and her brother had had a nervous breakdown. The patient was the second in a family of five. The domestic situation of the mother and father was unhappy and the patient had had to mind the other children. Subsequently she had worked with her father for five years. She had been married eleven years prior to the meeting and had three children. The marital relationship was unsatisfactory and *coitus interruptus* was practised. The patient had always had a jealous and argumentative disposition. She had been nervous, irritable and difficult for years, and quarrelled with the neighbours, police intervention being necessary at times. She was irritable with the children, threw saucepan lids around and was extremely jealous of her husband. Her frequent quarrels had made it necessary for them to leave several towns and the husband had had to apply for transfers. At the time of the meeting they were separated, as the husband could not get a house in Sydney where he was working. The patient said that life was not worth living and had talked of suicide, though she had never made any attempt on her own life. In the last three years the patient had consulted at least five doctors. She had left them one by one because she felt that they did not understand her or because she had fallen in love with them. She thought that she was excessively attractive to all men, and repeatedly asked: "Doctor, why are all men attracted to me physically?" She had other ideas of grandeur and believed that she had great cultural and literary talent. She continually wrote long and rambling letters to her doctor of the moment. On her admission to Broughton Hall she was found to be an emotional and jealous woman who related all her troubles to the sensuality of her husband. She denied the behaviour attributed to her, but admitted that she had fallen in love with "the dear doctor". The diagnosis was paraphrenia. The comment was made that this type of patient could get a doctor into endless trouble.

#### Tabes Dorsalis.

A female patient, aged forty-one years, had been married at the age of about twenty years. She had had a vaginal discharge after her marriage which lasted for seven months only; the husband was promiscuous. Her present condition was of four years' duration. She had a "sore skin" and the pressure of clothes caused great discomfort. She had had frequent falls, and suffered from sharp pains in the legs and in the forearms and sometimes in the face. Syphilis was diagnosed in June, 1947, although she had been under medical observation for some years. On examination of the patient the systolic blood pressure was 160 millimetres of mercury and the diastolic pressure was 96 millimetres. The pupils were unequal and eccentric and did not react to light. There were patchy areas of anaesthesia on the legs and trunk, while at the same time hyperaesthesia to light touch was present in the legs. The gait was ataxic and Rombergism was present; the knee jerks were absent. The cerebrospinal fluid was found to contain 22 cells per cubic millimetre and a slight amount of globulin; the Takata-Ara test and the Wassermann test both produced reactions, and the gold sol curve was "55544200000". The diagnosis of *tabes dorsalis* had been made. Attention was drawn to the absence of any secondary syphilitic signs, and it was suggested that the patient had probably been infected during her first marriage. The diagnosis should have been made much earlier. She had had a course of malaria treatment and was at the time of the meeting having a course of tryparsamide injections.

#### Subacute Combined Degeneration of the Cord.

The next patient was a female, aged forty-eight years, whose family history and early history were not significant. She had married at the age of twenty-eight years, but had separated from her husband after three years and had supported herself by working as a cutter. She was said to have been subject to tantrums and had been an inveterate gambler. She had undergone a salpingectomy twelve months after her marriage and a thyroidectomy ten years prior to the meeting. She had been referred to Broughton Hall because of loss of power in the legs. Some months previously she had been admitted to a general hospital, and her condition there was diagnosed as an anxiety neurosis; subsequently in another hospital it was diagnosed as hysteria. She had had a fall from a horse a week before her admission to hospital, and her symptoms were exaggerated. A relative said that for a considerable

time the patient had complained that her hands were sore, and she seemed to be always stumbling; over a period of twelve months her gait had progressively deteriorated. She had had attacks of hysterical screaming and had threatened suicide. She had gambled heavily and had been heavily in debt. Her arms became affected and she had difficulty in feeding herself. The patient said that she had had difficulty in walking for twelve months; she had trouble in doing her hair, and had burning feelings in her legs and tingling in her hands.

On examination of the patient her blood pressure was found to be 164 millimetres of mercury (systolic) and 100 millimetres (diastolic). Her gait was spastic and ataxic, the muscles of her hands were wasted and she had a tendency to wrist drop. The knee jerks were absent and the plantar reflexes were extensor in type on both sides. Investigation of the blood serum and the cerebro-spinal fluid gave negative results. The blood contained 5,920,000 erythrocytes per cubic millimetre and the colour index was 97. The patient had total achlorhydria.

It was pointed out that the circumstances of the patient were such as might reasonably be expected to lead to hysterical manifestations. However, objective examination left no doubt about the organic nature of her disability. She had in fact spastic ataxic paresis, which was suggestive of a combined lesion of the lateral and posterior columns of the cord, and the total achlorhydria confirmed the diagnosis of subacute combined degeneration of the cord. The absence of blood changes was not uncommon in this condition, and suggested that the sites of production of the blood-protective and neuro-protective factors in the stomach were not identical. It was also not uncommon to find in subacute combined degeneration evidence of peripheral neuritis, and in this patient neuritic changes probably accounted for the subjective pain, tingling and burning and for the wasting of the hand muscles. The patient had been treated with "Ventriculin", liver injections and vitamin B<sub>1</sub>. Her symptoms had not retrogressed, but on the other hand they had not advanced since her admission to hospital.

#### Dementia Paralytica.

A female patient, aged twenty-six years, had been admitted to hospital on October 28, 1947. She was the youngest of a family of seven, of which the other members were said to be healthy; the mother was alive and well and the father had died of a hematemesis. The patient had been married at the age of nineteen years and deserted by her husband two years prior to the meeting; she had two children, aged respectively six and two years. The patient's sister had noticed a change in her about eighteen months prior to the meeting; she began to talk loudly, lost interest in her appearance and became emotional and unstable. She complained of losing the use of her legs and used to "flap down"; she was bad-tempered, incontinent of urine and incapable of looking after herself and her children. In the middle of September, 1947, she was examined by a psychiatrist; her blood reacted to the Wassermann test. She had had four injections, presumably of arsenicals, before her admission to Broughton Hall. On her admission she was mentally retarded, her memory was poor and she was disorientated for time; she was rambling and incoherent and unable to give a history of herself. Her speech was slurred and stammering and her pupils were unequal; they reacted to light and accommodation. She had a gross tremor of the tongue of the "tremor" type and her hands were tremulous. The knee jerks were present and the plantar reflexes were flexor in type. At the time of the meeting the findings on examination of the cerebro-spinal fluid were not available. She had been inoculated with malaria and was undergoing treatment. The diagnosis was *dementia paralytica*, apparently acquired. One of the children had failed to react to the Wassermann test. The other was with the father and had not been examined.

#### Diffuse Sclerosis of Premotor Areas; Possible Involvement of the Corpus Callosum.

A female patient, aged sixty-one years, had had eight children, one having died. Her parents had died of old age, both at the age of about eighty years. The patient was reported to have been well until three years previously. The onset of symptoms was gradual; the family first noticed that she had difficulty in walking and then her voice became affected. She had been practically helpless for eighteen months prior to her admission to hospital on August 28, 1947. There was no history of apoplexy or of apocleticiform seizures. The patient had been actively employed until the onset of symptoms. After her admission to Broughton Hall she had an hæmoptysis, and X-ray examination revealed

tuberculous infiltrative changes in the apex of the left lung and pleural thickening at the base of the left lung. The patient was a debilitated woman looking older than her years. The blood pressure was 150 millimetres of mercury (systolic) and 90 millimetres (diastolic). The sense of smell, visual acuity and sensation in general were difficult to examine because of the patient's speech disability; her oculomotor functions were intact. The pupils were small and slightly irregular and reacted slowly to light; the accommodation reaction was not observed. The motor functions of the fifth cranial nerve were affected; movements of the jaw were weak and mastication was difficult. The sensory functions of the fifth cranial nerve were intact as far as could be determined. Hearing was good and the vestibular functions were not tested. The patient protruded her tongue in the mid-line; the palate moved very little during phonation, but there was no deviation. The corneal, conjunctival and palatal reflexes were present. Speech was grossly anarthric and unintelligible, but the patient could phonate. The general sensory functions were preserved as far as could be determined in view of the patient's difficulty in cooperating. She had lost control of her sphincters. Hypertonus of the upper and lower extremities was present—passive movement excited hypertonic rigidity. For example, when the knee was passively flexed the lower extremities became rigid and the body could be raised *en masse*. The gait was feeble and spastic, and the patient could walk only with assistance. The toes tended to adopt an attitude of dorsiflexion. The abdominal reflexes were active; the biceps and triceps reflexes were active; the knee jerks could not be elicited, on account of the rigidity of the extremities, which she could not voluntarily relax. Both plantar reflexes were flexor in type. She exhibited a grasp reflex in both hands and feet—her hands always tended to grope after objects. Her movements generally were clumsy and ill-directed. Investigation of the blood serum and the cerebro-spinal fluid gave negative results; there were no changes in the cerebro-spinal fluid suggestive of tuberculous infection of the central nervous system. No papilloedema and no signs of intracranial tension were detected.

The comment was made that the symptoms suggested pseudo-bulbar palsy, but not of usual type, in view of the absence of pyramidal involvement and of a history of apoplexy. The motor disability was probably of the nature of apraxia. The mental state could not be determined with any degree of accuracy, but probably considerable dementia was present. The provisional diagnosis was *diffuse sclerosis of the premotor areas* with possible involvement of the *corpus callosum*. The groping and grasping were probably release phenomena.

#### Depressive State of Psychogenic Origin.

The next patient was a male, aged about thirty-six years, a labourer, whose mother had committed suicide at the age of fifty-five years; the patient was the third in a family of five. He had done labouring work all his life, and had remained for many years in the same position, being well thought of; he was a good mixer, had a bright disposition, was fond of dancing and played the banjo at parties; he had taken an active part in sport when younger, and still liked to watch sport on Saturday afternoons. Nine months previously he had had an accident at work—his arm had been badly fractured in three places; he spent three weeks in Saint Vincent's Hospital. He became depressed and could not shake off his depression; he had threatened suicide and been sent to the Reception House, where he stayed for days, but he was not certified insane. He went back to work, but found that he could not concentrate and that he felt tired all the time. He slept badly and had severe limitation of pronation as a result of the accident. The trade unions were taking the case up and claiming compensation. Six weeks after the accident the patient felt fairly well; the precipitating factor of his depression was a letter from the hospital asking him to attend and have his arm manipulated. In his unsettled emotional state it seemed to him that this meant that his arm would have to be amputated, and he had never been the same since. On his admission to Broughton Hall he was depressed and restless, contemplating suicide and feeling that he might injure others. Electro-convulsive treatment was in progress at the time of the meeting and the patient's condition was much improved.

#### Chorea of Huntington Type.

The next patient shown was a male, suffering from chronic progressive chorea of Huntington type without any known hereditary factor. The patient had had an accident at work and maintained that the condition had commenced

after the accident; he was claiming compensation, and at the time of the meeting judgement had not been delivered. The patient maintained, and was obviously well conditioned to maintaining, that he had been perfectly well until the accident. He stated that a few days afterwards he had experienced difficulty in rolling a cigarette, and from then onwards the disease had progressed. As the case was *sub judice*,<sup>1</sup> no opinion could be expressed on that aspect. The comment was made that it was interesting to note that many sufferers from Huntington's chorea in the early stages were not aware that they had the disease and might actually stoutly deny it. Wechsler had made the following statement: "I have seen almost classical types of Huntington's chorea, including mental deterioration, in patients who showed neither familial nor hereditary traits."

## Medical Societies.

### MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held on June 11, 1947, at the Children's Hospital, Melbourne, the acting chairman, DR. H. BOYD GRAHAM, in the chair. Part of this discussion appeared in the issue of March 20, 1948.

#### Spontaneous Mediastinal Emphysema.

DR. V. L. COLLINS read a paper entitled "Mediastinal Emphysema" (see page 614) and showed a patient suffering from spontaneous mediastinal emphysema.

DR. R. L. FORSYTH said that he proposed to tell of one of his famous mistakes; he was the only person who ever made mistakes. The patient was not a child, but the case was one of spontaneous pneumothorax. The patient was a well-known sprinter, who found that he was unable to run as freely as previously. Dr. Forsyth said that he found diminished air entry over one lung. The radiologist reported pulmonary tuberculosis, and the patient was ordered to give up running. However, he ran the following year, and with great success. No doubt the appearances were due to spontaneous pneumothorax, and not due to tuberculous infection. Spontaneous pneumothorax was more common in athletes than was at first thought.

DR. ALAN PENINGTON said that surgical emphysema frequently followed thoracoscopy. Not infrequently in these cases, after coughing, air was present above the sternal notch and in the neck. After pneumoperitoneum air might pass along the posterior mediastinum into the neck, but in the anterior mediastinum the air appeared to become loculated, and extended into the neck only from the posterior mediastinum. Dr. Penington said that he was not convinced that pulmonary interstitial emphysema was a frequent forerunner of spontaneous pneumothorax, and that most of these cases were due to rupture of emphysematous bullae, which were frequently seen on thoracoscopy, although not easily demonstrated in autopsy work. Dr. Penington said that he found it hard to explain how air from the anterior mediastinum could pass into the pleural sac, as the mediastinal pleura was usually extremely tough.

DR. GUY SPRINGTHORPE asked whether in some cases air embolism had developed. He had had a painful experience whilst giving an anæsthetic for the removal of a piece of rabbit's bone in the œsophagus. A rupture of lung tissue developed, and the patient became unconscious and was subsequently found to have unilateral pneumothorax and complete hemiplegia. In the French literature, the establishment of pneumothorax for tuberculosis was a common procedure, and air embolism was not very uncommon. Dr. Springthorpe also asked if there was any likelihood of recurrence in this case.

Dr. Collins, in reply, said that he agreed with Dr. Penington on the usual manner in which spontaneous pneumothorax began. Most evidence favoured the emphysematous bullae theory, but in a certain percentage of cases pulmonary interstitial emphysema was the cause. In answer to Dr. Springthorpe, Dr. Collins said he had not met with air embolism in discussion of induced pneumothorax. Simmonds had reported a number of cases of embolism after pneumoperitoneum, air apparently travelling by the lumbar veins directly to the brain, by-passing the heart. A similar phenomenon apparently occurred with embolism in the puerperium. In many cases mediastinal emphysema had

recurred. Most people felt that some other factor was present, such as a congenital weakness; otherwise, every pregnant woman and every asthmatic would suffer this complication.

## Post-Graduate Work.

### THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

#### PROGRAMME FOR JUNE, 1948.

THE Melbourne Permanent Post-Graduate Committee announces the following programme for June, 1948.

#### Course in Endocrine Disorders, including Diabetes.

The following demonstrations, suitable for candidates for the M.D. Part II and M.R.A.C.P., will be conducted at 2 p.m. on Tuesdays and Thursdays under the direction of Dr. Keith D. Fairley: June 1, at the Royal Melbourne Hospital, "Disorders of the Thyroid Gland", Dr. K. D. Fairley; June 3, at the Royal Melbourne Hospital, "Disorders of the Pituitary Gland", Dr. John Bolton; June 15, at the Alfred Hospital, "Disorders of the Adrenal Glands", Dr. R. Andrew; June 17, at the Royal Melbourne Hospital, "Diabetes Mellitus", Dr. W. McI. Rose; June 22, at the Women's Hospital, "Endocrinology in the Female", Professor J. W. Johnstone; June 24, at Saint Vincent's Hospital, "Disorders of the Parathyroid Glands", Dr. M. Biggins.

#### Classes at the University of Melbourne for Higher Degrees and Diplomas.

Classes for Part I of the M.D., M.S., D.O., D.G.O., D.D.R., D.T.R.E. and D.A., and in physics for Part II of the D.T.R.E., which commenced in March, will be continued.

#### Demonstration at Geelong Hospital.

Mr. G. Swinburne will conduct a demonstration on "The Acute Ear" at the Geelong Hospital on Wednesday, June 9, at 8.30 p.m. Dr. N. W. Morris, "Belleville", Ryrie Street, Geelong, will make enrolments for this course.

#### Week-End Course at Mooroopna Hospital.

A course will be conducted at Mooroopna on June 19 and 20 as follows:

Saturday, June 19: 2.30 p.m., "Surgery of the Stomach", Mr. D. R. Leslie; 8 p.m., "The Failing Heart", Dr. H. B. Kay.

Sunday, June 20: 10 a.m., "The Rehabilitation of Midwifery Patients", Mr. D. F. Lawson; 2 p.m., "Some Recent Advances in Pædiatrics", Dr. V. L. Collins.

The fee for this course is £2 2s. and enrolments should be made with Dr. B. S. Schloeffel, Shepparton.

#### Enrolments.

Inquiries and enrolments for courses other than those at country centres should be made at the committee's office, Melbourne.

#### INTENSIVE REFRESHER COURSE FOR GENERAL PRACTITIONERS.

The date of the intensive refresher course for general practitioners has been altered, and it will now be held a week later, from September 6 to 17, to coincide with the visit of Professor Spence.

#### REPRINTS OF PROFESSOR F. M. BURNET'S LECTURES.

Reprints of Professor F. M. Burnet's lectures, "The Background of Infectious Diseases in Man", are now available from the committee, price 7s. 6d.

### THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

#### Course in Advanced Medicine.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a course in advanced medicine suitable for M.R.A.C.P. candidates will be conducted for a period of twelve weeks from June 15 to September 3, 1948, the fee for which will be £31 10s. The

<sup>1</sup> Compensation was subsequently refused.

programme has been arranged to take place almost exclusively in the afternoons from approximately 2 to 5 p.m. on five days per week. Extra sessions may be arranged on certain Saturdays. The course will include the following: (i) didactic lectures on the more obscure aspects of internal medicine, designed to supplement the students' reading; these will cover the various systems in turn; (ii) lectures and tutorials in electrocardiography; (iii) ward rounds and demonstrations at the principal metropolitan hospitals approximately twice a week; (iv) library seminars at which recent literature on set subjects will be discussed; (v) demonstrations of the *fundus oculi*; (vi) lecture-demonstrations in physiology and biochemistry and discussions on applied physiology; (vii) lecture-demonstrations in pathology and haematology; (viii) demonstrations of the application of radiological methods of diagnosis to medical diseases; (ix) demonstrations of psychiatric conditions; (x) the exhibition of selected medical films; (xi) portions of the annual post-graduate course of interest to students in advanced medicine.

The supervisor of the course, Dr. Selwyn G. Nelson, will conduct tutorials on selected subjects, and students may discuss with him any problems arising in the course of their work.

It is expected that students will devote a considerable time to general reading, both of textbooks and of current medical literature. The object of this course is to provide assistance and guidance for the serious student of internal medicine. It is desirable that students should have had considerable clinical experience in hospital and/or medical practice before considering themselves prepared to take examinations for higher medical degrees or diplomas.

Applications to attend whole or portion of the course in advanced medicine should be in the hands of the Course Secretary, Post-Graduate Committee in Medicine, 131, Macquarie Street, Sydney, not later than May 24, 1948.

## Naval, Military and Air Force.

### APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 57 and 65, of April 8 and 22, 1948.

#### PERMANENT NAVAL FORCES OF THE COMMONWEALTH (SEA-GOING FORCES).

##### Retired List.

**Termination of Appointment.**—The appointment of Frank Frederick Coffey as Surgeon Lieutenant-Commander is terminated, dated 29th February, 1948.

#### AUSTRALIAN MILITARY FORCES.

##### Interim Army.

##### Australian Army Medical Corps.

VX504168 Captain (Temporary Lieutenant-Colonel) P. Jones relinquishes the temporary rank of Lieutenant-Colonel and is transferred to the Reserve of Officers (Australian Army Medical Corps) (3rd Military District), 5th February, 1948.

TX6489 Captain (Temporary Major) R. W. Henning relinquishes the temporary rank of Major and is transferred to the Reserve of Officers (Australian Army Medical Corps) (2nd Military District), 19th February, 1948.

VX96327 Captain E. L. G. Beavis is transferred to the Reserve of Officers (Australian Army Medical Corps) (3rd Military District), 25th February, 1948.

16th *Camp Hospital*.—NX208039 Captain D. J. Stanger is transferred to the Reserve of Officers (Australian Army Medical Corps) (2nd Military District), 17th February, 1948.

20th *Field Ambulance*.—NX206864 Captain (Temporary Major) W. Inglis relinquishes the temporary rank of Major and is transferred to the Reserve of Officers (Australian Army Medical Corps) (2nd Military District), 17th February, 1948.

To be *Temporary Major*, 29th January, 1948.—NX506173 Captain M. A. Jackson.

##### Reserve Citizen Military Forces.

##### Australian Army Medical Corps.

2nd *Military District*.—Lieutenant C. McNerney (*née* Dewar) is placed upon the Retired List (2nd Military District) with permission to retain her rank and wear the prescribed uniform, 1st March, 1948.

#### ROYAL AUSTRALIAN AIR FORCE.

##### Reserve: Medical Branch.

Ex-Squadron Leader Eric Francis Langley (252868) is appointed to a commission with the temporary rank of Squadron Leader, 20th February, 1948.

The appointment of Flight Lieutenant F. J. Kenny (254851) is terminated at his own request, 17th February, 1948.

## Honours.

### THE ORDER OF THE HOSPITAL OF ST. JOHN OF JERUSALEM.

THE following admissions to or promotions in the Order of the Hospital of St. John of Jerusalem have recently been made among medical officers in Australia.

**Promoted to the Grade of Knight.**—Dr. H. F. J. Norrie, New South Wales; Brigadier W. W. S. Johnston, O.B.E., D.S.O., Victoria; Colonel H. H. E. Russell, O.B.E., South Australia.

**Promoted to the Grade of Dame.**—Dr. Frances C. B. McKay, New South Wales.

**Promoted to the Grade of Commander (Sister).**—Dr. K. Ardill Brice, O.B.E., New South Wales.

**Promoted to the Grade of Commander (Brother).**—Major-General F. A. Maguire, C.M.G., D.S.O., V.D., New South Wales; Lieutenant-Colonel E. A. H. Russell, South Australia; Colonel Allan R. S. Vickers, Queensland.

**Admitted in the Grade of Officer (Brother).**—Colonel A. M. McIntosh, D.D.M.S., New South Wales; Colonel G. G. L. Stening, New South Wales; Dr. E. MacArthur Sheppard.

**Admitted in the Grade of Serving Brother.**—Dr. Alfred C. Ternes, New South Wales; Dr. Leslie S. Woods, New South Wales; Dr. A. M. Purves, New South Wales; Dr. W. E. George, New South Wales; Dr. E. H. Evans, New South Wales; Dr. L. H. Foy, New South Wales; Dr. R. S. Steel, New South Wales; Dr. C. W. Flynn, New South Wales; Dr. H. F. Summons, M.B.E., Victoria; Dr. H. H. Hurst, South Australia.

## Correspondence.

### DYSOSTOSIS CRANIO-FACIALIS.

SIR: I have just received THE MEDICAL JOURNAL OF AUSTRALIA of January 17, 1948, containing the interesting letter and photographs from Dr. E. A. Joske. The condition illustrated can be confidently diagnosed as *dysostosis cranio-facialis* (Cruzon's disease). The following features are typical: familial incidence, "parrot-shaped" nose, prognathism, exophthalmos and divergent strabismus. The cause is an abnormality of growth of the skull bones, possibly related to other diseases like oxycephaly and acrocephalo-syndactyly with which it may be confused. The inheritance may be as a pure or irregular dominant, or familial incidence may be quite invident. The disease is rare, but sufficiently known to be described briefly in current neurological textbooks. I have seen two cases in England recently, one adult and a youth aged thirteen years. The adult is an imbecile and an inmate of a mental hospital, but the youth is of normal intelligence and was seen as an out-patient of an ophthalmic hospital. In these families no similar conditions are known, the patients are unrelated and there is no consanguinity in the parents.

#### Case Reports.

CASE I.—H.J.A., a male, aged thirteen years, born one month prematurely, birth weight three and a half pounds. His large head was noticed at birth and he was thought to be hydrocephalic and blind. For two and a half years he lay dormant, but then began to thrive. When examined, aged thirteen years, his height was four feet nine inches and he weighed 73 pounds. He has the typical facies with parrot-shaped nose, prognathism and exophthalmos but no divergent strabismus. His right vision is  $\frac{1}{12}$  and left vision  $\frac{1}{6}$  when wearing a low myopic correction. Ocular fundi and media show no abnormality. He was eleventh in his class of forty-nine at an average standard for his age.

His only disability is his appearance. He wears spectacles constantly, mostly to disguise his eyes.

CASE II.—J.L., a male, aged forty-four years. He was born one month prematurely, being the first of three children. One sister is said to be alive and normal, but the other died when aged two years, the cause being unknown. During infancy he was observed to be suffering from mental deficiency and spastic diplegia, both of which have persisted. He shows the typical features of large skull base, wide shallow orbits with exophthalmos and divergent strabismus, parrot-shaped nose and prognathism. The ocular fundi show small irregular atrophic and pigmented patches at each macula similar to those seen in some cases of senile macular degeneration. The visual acuity cannot be tested, but there are no suggestions of any optic atrophy.

#### Comment.

In the family described by Dr. E. A. Joske the inheritance is definite and shows at least some degree of dominance. The risks of further children being affected must therefore be considerable. That Mr. H. had a brother who was affected by some form of skeletal growth deficiency is interesting. The whole family tree appears worthy of publication if the members can be traced. Several references can be found in "Modern Trends in Ophthalmology", edited by Ridley and Sorsby, 1939, Butterworth, London.

Yours, etc.,

RONALD F. LOWE, D.O.M.S. (London),  
F.R.A.C.S., F.R.C.S. (England).

University of London,  
Institute of Ophthalmology,  
April 17, 1948.

## Obituary.

### ALBERT EDWARD PLATT.

A CONTRIBUTOR who wishes to remain anonymous has sent the following appreciation of the late Dr. Albert Edward Platt.

The sudden death on Thursday, April 8, 1948, of Dr. A. E. Platt, Director of the Institute of Epidemiology and Preventive Medicine and Director of the McIlraith Department of Pathology at the Prince Henry Hospital, came as a shock to his friends and colleagues. He is survived by his widow and son.

After graduating M.B., B.S. at the University of Sydney he spent some years in New Guinea. It was here that he built up his first laboratory from kerosene tins and odds and ends. This ability to design and build apparatus with whatever material was available was indeed a gift. Dr. C. H. Kellaway recently described him as a "master of improvisation". His intense interest in the laboratory as a tool in the analysis of problems relating to infectious disease eventually led him to the London School of Hygiene and Tropical Medicine, where he was fortunate enough to study under and become a close acquaintance of the late Professor W. W. C. Topley. Platt absorbed some of the spirit and determination of this great teacher, and on his advice took the positions of bacteriologist to the Royal Adelaide Hospital and lecturer in bacteriology to the University of Adelaide in 1935. He later became Deputy Director of the Institute of Medical and Veterinary Science, Adelaide.

His keenness on fundamental medical research and his ability as a teacher of both science and medical students were soon recognized, and he was appointed Professor of Bacteriology at the University of Adelaide in 1938. Platt then started a full-time university course in bacteriology for science students. His early hopes have now been realized, as his students are doing first-class work in almost every State in this country.

Having pioneered this particular field, Platt resigned from the chair of bacteriology at Adelaide to take up the position of bacteriologist at the Prince Henry Hospital, New South Wales, and was soon appointed Acting Director of the McIlraith Department of Pathology. His interest in preventive medicine and epidemiology had been increasing for some time and became manifest in his research work and discussions. As a result of his ideas and rich experience in this field the Institute of Epidemiology and Preventive Medicine was established at the Prince Henry Hospital and he was appointed director in 1946, which position he held until his death. A few months ago his international reputation was recognized when he was appointed as an abstract writer for *Excerpta Medica*.

His enthusiasm, sincerity and sound advice will be remembered and appreciated by all those who studied under

him. He possessed a retiring disposition and his comments were concise and to the point as he had a remarkable ability to present basic facts clearly. He expected of his students a standard of work and thought that taxed their efforts to the utmost. He will be remembered by those who knew him well for his humility and gentleness and as a man who possessed a deep and great understanding of fundamental biological phenomena. One with the great scientists of all time, he would fight to the last to defend a scientific principle and the right of an individual to investigate the problems of nature.

#### Some Publications of the Late Albert Edward Platt.

- "The Viability of Bact. Coli and Bact. Aerogenes in Water: A Method for the Rapid Enumeration of these Organisms", *Journal of Hygiene*, Volume XXXV, 1935, page 437.
- "The Relationship of the Complement Fixation Optimum to the Agglutination Optimum", *The Australian Journal of Experimental Biology and Medical Science*, Volume XIV, 1936, page 101.
- "Investigations into the Nature of the Condition known as 'Floating Yolk' in Eggs", *The Australian Journal of Experimental Biology and Medical Science*, Volume XIV, 1936, page 107.
- "Brucella Infections: The Frequency of Agglutinins for Brucella Abortus in the Population at Large", *THE MEDICAL JOURNAL OF AUSTRALIA*, February 22, 1936, page 268.
- "A Serological Study of Hemophilus Influenzae. I", *Journal of Hygiene*, Cambridge, Volume XXXVII, 1937, page 93.
- "Salt Optima in Agglutination", *The Australian Journal of Experimental Biology and Medical Science*, Volume XVI, 1938, page 275.
- "A Serological Study of Hemophilus Influenzae. II. Two Serologically Active Protein Fractions Isolated from Pfeiffer's Bacillus", *The Australian Journal of Experimental Biology and Medical Science*, Volume XVII, 1939, page 19.
- "Observations on the Role of Sodium Chloride in the Agglutination of Bacteria by Antibody", *The Australian Journal of Experimental Biology and Medical Science*, Volume XIX, 1941, page 57.
- "Improved Equipment for Field Bacteriological Laboratories", *Australian Military Forces*, 1941.

### WILLIAM MACDONALD HELSHAM.

We regret to announce the death of Dr. William Macdonald Hesham, which occurred on April 27, 1948, at Narrabeen, New South Wales.

### MARCEL URBAIN CRIVELLI.

We regret to announce the death of Dr. Marcel Urbain Crivelli, which occurred on May 1, 1948, at Melbourne.

### CHARLES STANLEY BLAKE LANGDON.

We regret to announce the death of Dr. Charles Stanley Blake Langdon, which occurred on May 1, 1948, at Melbourne.

### DAVID GIFFORD CROLL.

We regret to announce the death of Dr. David Gifford Croll, which occurred on May 4, 1948, at Brisbane.

### ROY VESCYS GRAHAM.

We regret to announce the death of Dr. Roy Vescys Graham, which occurred on May 4, 1948, at Sydney.

## Congresses.

### INTERNATIONAL CONGRESS ON INDUSTRIAL MEDICINE.

THE ninth International Congress on Industrial Medicine will be held at London under the aegis of the *Commission internationale pour la médecine du travail* on September 13-17, 1948. The congress will work in six sections devoted

to the following aspects of industrial medicine: "Social Aspects", "Environment", "Nursing", "Clinical", "Practice" and "Special". Arrangements have been made for members to visit a representative selection of rehabilitation centres, clinics, hospitals *et cetera*, both in London and in extra-metropolitan areas. The official languages will be French and English and the congress fee will be £3 (sterling). The organizing secretary is Miss G. B. Mawdesley, Room 501, Garden Court Wing, B.M.A. House, Tavistock Square, London, W.C.1.

#### AN UNUSUAL DISTINCTION FOR DR. CHRISTOPHER ARTHUR GRIFFITH.

An unusual distinction has been gained by Dr. Christopher Arthur Griffith, of Harkaway, near Berwick, Victoria. Dr. Griffith qualified in England in 1886 and became a member of the Royal College of Surgeons of England. He has practised for many years in Victoria, and at the present time lives in semi-retirement at Harkaway. He has been granted the diploma of Fellow of the Royal College of Surgeons of England by the application of the special rule making members of long service eligible for fellowship. We extend our congratulations to Dr. Griffith.

### Medical Appointments.

Dr. J. J. L. McDonald has been appointed medical officer, Department of Public Health, New South Wales.

Dr. A. G. Campbell has been appointed assistant medical superintendent, Surgical Branch, Royal Adelaide Hospital, Adelaide.

The undermentioned appointments have been made at the Royal Alexandra Hospital for Children, Camperdown, Sydney: Honorary Assistant Physicians, Dr. M. L. Edwards and Dr. D. G. Hamilton; Honorary Relieving Assistant Physicians, Dr. J. M. Alexander, Dr. G. M. Blaxland, Dr. C. W. G. Lee and Dr. S. E. J. Robertson; Honorary Dermatologist, Dr. A. J. P. Chapman; Honorary Assistant Dermatologist, Dr. R. F. A. Becke; Honorary Relieving Assistant Dermatologist, Dr. R. H. Kaines; Honorary Assistant Ear, Nose and Throat Surgeon, Dr. R. E. Dunn; Honorary Relieving Assistant Ear, Nose and Throat Surgeon, Dr. Patricia R. Davey.

Dr. W. F. Salter, Deputy Superintendent of the Northfield Mental Hospital, has been appointed Deputy Superintendent at the Parkside Mental Hospital and also Deputy Superintendent of the Hospital for Criminal Mental Defectives, pursuant to the *Mental Defectives Act*, 1935 to 1941, of South Australia.

Dr. J. S. T. T. Hill has been appointed honorary clinical assistant to the Medical Section of the Royal Adelaide Hospital, Adelaide.

### Books Received.

"Conference on Liver Injury: Transactions of the Fifth Meeting, September 26-27, 1946, New York", edited by Dr. F. W. Hoffbauer; New York: Josiah Macy Junior Foundation. 9" x 6", pp. 128, with illustrations. Price: \$2.25.

"Liver Injury: Transactions of the Sixth Conference, May 1 and 2, 1947, New York", edited by F. W. Hoffbauer; New York: Josiah Macy Junior Foundation. 9" x 6", pp. 74, with illustrations. Price: \$2.00.

"The U.F.A.W. Handbook on the Care and Management of Laboratory Animals: With an Appendix on Statistical Analysis", edited by Alastair N. Worden, M.A. (Cantab.), B.Sc. (London), M.R.C.V.S., A.R.I.C., with a foreword by Professor T. Dalling, M.A., M.R.C.V.S., F.R.S.E.; 1947. London: Baillière, Tindall and Cox. 9½" x 6½", pp. 384, with illustrations. Price: 31s. 6d.

"Treatment in General Practice", by Harry Sturgeon Crossen, M.D.; Sixth Edition; 1948. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9½" x 6½", pp. 1146. Price: £4 0s. 6d.

"Operative Gynecology", by Harry Sturgeon Crossen, M.D., and Robert James Crossen, M.D.; Sixth Edition; 1948. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 10" x 7", pp. 1022, with many illustrations, some of them coloured. Price: £5 12s. 6d.

"Occupational Medicine and Industrial Hygiene", by Rutherford T. Johnstone, A.B., M.D.; 1948. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9½" x 6½", pp. 604, with many illustrations, some of them coloured. Price: 75s.

"Introduction to Physiology", by W. H. Newton, D.Sc., M.D.; 1948. London: Edward Arnold and Company. 7½" x 5", pp. 284, with illustrations. Price: 7s. 6d.

"The 1947 Year Book of Physical Medicine", edited by Richard Kovács, M.D.; 1948. Chicago: The Year Book Publishers Incorporated. 7" x 4½", pp. 432, with illustrations. Price: \$3.75.

### Diary for the Month.

May 17.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.

May 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.

May 19.—Western Australian Branch, B.M.A.: General Meeting.

May 20.—New South Wales Branch, B.M.A.: Clinical Meeting.

May 20.—Victorian Branch, B.M.A.: Executive Meeting.

May 25.—South Australian Branch, B.M.A.: Branch Meeting.

May 25.—New South Wales Branch, B.M.A.: Ethics Committee.

May 26.—Victorian Branch, B.M.A.: Council Meeting.

May 27.—New South Wales Branch, B.M.A.: Branch Meeting.

### Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

**New South Wales Branch** (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phoenix Mutual Provident Society.

**Victorian Branch** (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

**Queensland Branch** (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

**South Australian Branch** (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

**Western Australian Branch** (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

### Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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